OCT 30 1925

VOL. LIV. NOVEMBER, 1925 No. 6.

ARCHIVES OF OPHTHALMOLOGY

Medical Lib

FOUNDED IN 1869 BY HERMAN KNAPP

EDITED BY

ARNOLD KNAPP AND WARD A. HOLDEN

NEW YORK

VISION IN LIGHT OF REDUCED INTENSITY
CARSTEN EDMUND AND H. ULRIK MÖLLER

THE TREATMENT OF TABETIC OPTIC ATROPHY
E. L. ZIMMERMANN

SUBNORMAL ACCOMMODATION . . . ALEXANDER DUANE

EXCRESCENCES OF THE CENTRAL AREA OF DESCEMET'S MEMBRANE . . . . . DANIEL B. KIRBY

DACRYOPS. A REPORT OF THREE CASES . OTTO LANDMAN

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE
DAVID H. WEBSTER

DISCUSSION ON "THE FUNDUS OCULI IN GENERAL MEDICINE" THE MEDICAL SOCIETY OF LONDON . . H. DICKINSON

(Complete contents inside)

G. P. PUTNAM'S SONS
2 WEST 45TH ST., NEW YORK

PUBLISHED BIMONTHLY SUBSCRIPTION, SIX DOLLARS A YEAR

Entered at the Post-Office, New Rochelle, N. Y., as Second-Class Mail Matter

NOTICE TO CONTRIBUTORS

The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with the publication of their contributions.

1. Authors will receive gratuitously twenty-five reprints of their articles. If a greater number is desired,—**notice of which should be given at the head of the manuscript, or at the latest on the first proof,**—only the additional cost of press-work and paper will be charged to the author. The **number so ordered should be inclusive of the gratuitous copies.**

2. In preparing manuscript for the compositor it is requested that the following rules be adhered to:

a. Write on one side of the paper. Type-written MS. is preferred.

b. Write without breaks, *i. e.*, do not begin each sentence on a new line. When you want to begin a new line or paragraph at a given word, place before it in your MS. the sign ¶.

c. Draw a line along the margin of such paragraphs as should be printed in smaller type—for instance, all that is clinical history in reports of cases, etc.

d. Words to be printed in *italics* should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times; **antique** type should be so marked.

e. Let the title of your paper indicate its contents. If it is a general title, for instance, Clinical Contributions, mention the subject of each special communication,—for instance: CASE I. Sarcoma of Iris; CASE II. Exostosis of Frontal Sinus, etc. These special titles will appear in the table of contents of each number and each volume, under the heading of the general title, so that they will not be overlooked.

f. Illustrations should be carefully drawn on separate sheets.

3. Authors receive proofs for revision, which they should correct and return without delay. We beg, however, to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them to make, if possible, no alterations at all in their MSS., or, at least, to limit them to what is of essential importance.

When sending manuscripts for the ARCHIVES OF OPHTHALMOLOGY, please address the editor, Dr. ARNOLD KNAPP, 10 East 54th St., New York City.

ARCHIVES OF OPHTHALMOLOGY.

VISION IN LIGHT OF REDUCED INTENSITY.

By Dr. CARSTEN EDMŬND and H. ULRIK MÖLLER, Copenhagen.

From the Eye Clinic of the University of Copenhagen.
Chief: Professor Tscherning.

(With two figures on Text-Plate XVIII. and six in the text.)

PHOTOMETRIC GLASSES. In order to grade the intensity of light easily, a series of gray glasses, designated as *photometric glasses*, have been manufactured according to Professor Tscherning's specifications. They are, as far as possible, neutral-gray glasses, that is to say, they absorb all the rays of the spectrum to an equal degree. These glasses are numbered from 1 to 10, in such a manner that number 1 transmits $\frac{1}{10}$ of the light, Nr. 2 $\frac{1}{100}$, Nr. 3 $\frac{1}{1000}$, and so on. The unit is called a *photoptry* (*Ph.*). Thus, a glass of n photoptries transmits $\frac{1}{10^n}$ of the light. The advantage of employing such a scale is that the glasses may be added in the same manner as spectacle-glasses measured in dioptries. If, for instance, Ph. 2 is combined with Ph. 1, the effect of the combination becomes equal to that of Ph. 3. Just as it proved insufficient for dioptry glasses to have whole numbers only, the photoptry spectacle box[1] contains, in addition to the glasses already mentioned, the numbers 0.25, 0.50, and 0.75. Thus,

[1] The contrivances mentioned in this article are manufactured by N. Fischer, 32, Aaboulevard, Copenhagen N. U. S. A. Agency, A. Myers, 7313 Pingree Street, Chicago.

in combining these with the whole numbers, the following scale is obtained:

Nr. of glass:	0.25	0.50	0.75	1.00	1.25	1.50	1.75	2.00
	2.25	2.50	2.75	3.00 etc.				
Transparence:	0.56	0.32	0.18	0.10	0.056	0.032	0.018	0.010
	0.0056	0.0032	0.0018	0.0010 etc.				

The glasses are circular and have the same diameter as ordinary spectacle-glasses (38*mm*). The absorption takes place by a gelatin plate which is tinted with anilin coloring matter (neutralgrau—neutral-gray—from Meister Lucius & Brüning, Höchst a M, Germany). The gelatin plate is set in Canada balsam between two glass plates.[1]

The glasses are intended chiefly for the examination of patients. They may, of course, be used like spectacle-glasses, and it is possible to combine them with dioptry glasses by replacing the plane glass, between which the gelatin plate is placed, by plano-convex (-concave) lenses. That will however scarcely prove practical, because, in the course of time, most anilin colors are influenced by light. Therefore, it is recommended that the box in which the glasses are kept be closed when the glasses are not in use. It would however be very convenient if the opticians would introduce Tscherning's scale for the ordinary gray glasses.

---

[1] The exactness with which the Ph. glasses can be produced cannot be compared with the almost mathematical exactness with which the diopter glasses are produced. Ph. 2 contains twice as much coloring matter as Ph. 1, it should, therefore, be equivalent to Ph. 1+Ph. 1. But even a gelatin film without coloring matter absorbs a little light, and the same applies to the glass plates between which the gelatin plate is set. Moreover, there is the additional loss of light caused by the absorption from the glass plates.

That is the reason why the combination as a rule will be somewhat darker than a single glass. These slight losses do not matter with the higher numbers, but on the other hand, it is more difficult to produce the powerful glasses with sufficient exactness, for the slightest deviation in the thickness of the gelatin film will cause a rather considerable change in the power of the glasses.

In regard to the question to what degree the glasses may be designated as neutral-gray, that depends greatly on the coloring matter supplied by the chemical works, owing to the fact that it is not always of the same quality.

ILLUSTRATING THE ARTICLE OF DR. CARSTEN EDMUND AND H. ULRIK MÖLLER ON "VISION IN LIGHT OF REDUCED INTENSITY."

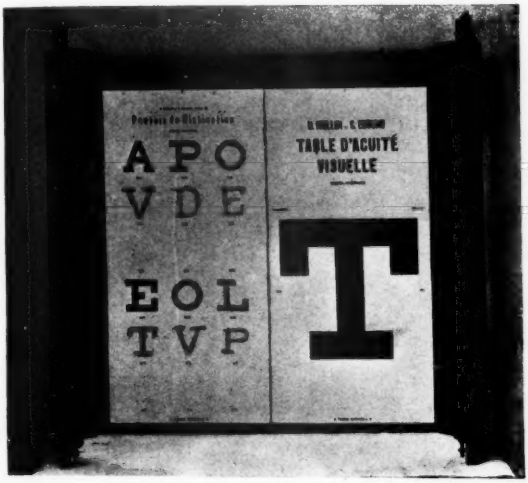


FIG. 1A

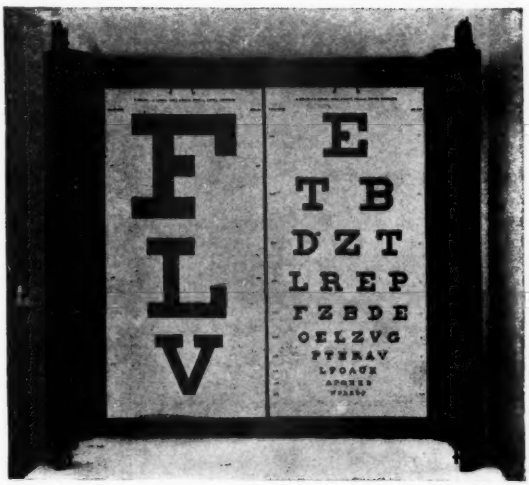


FIG. 1B

The Ph. spectacle box contains a frame, by means of which the glasses may be placed light-proof in front of the eye. A little cotton may be placed on the inside of the edge of the frame in order to make it fit better around the orbit.

Scale of brightness. According to Tscherning's latest suggestion, the degree of brightness (c) of an object is determined by the strongest Ph. glass through which a maximally adapted eye is just able to see it. One is considered to be maximally adapted after having worn Ph. 10 placed light-proof before the eye for half an hour. Under such conditions, most persons will just be able to see a square of white paper, $27.5mm^2$, placed at one meter's distance and illuminated by a standard candle, through Ph. 5. The degree of brightness of this paper is therefore put at $c=5$. If the candle is replaced by a 10 candle lamp, the degree of brightness of the paper becomes $c=6$, while a 100 candle lamp gives a brightness of 7, and a 1000 lux argenta lamp a degree of brightness of 8. The degree of brightness of the flame of a standard candle is 10, most normal persons just being able to perceive the flame through Ph. 10, when the eye is at its maximum adaptation. The unit of brightness is the faintest light which a maximally adapted eye is able to perceive. It corresponds to $\frac{1}{10^{10}}$ of the brightness of the candle flame, or to the brightness of a sheet of white paper illuminated by a standard candle at a distance of $316m$. This brightness corresponds to zero of the scale. That the brightness of an object is $=5$ means that its brightness is 10^5 times as great as the unit.[1]

---

[1] As the statements of otherwise normal persons are not all perfectly alike, it is necessary, if one wishes to use Ph. glasses for measurements of this kind, to commence by measuring the degree of brightness of the square of white paper illuminated by a standard candle at one meter's distance. If one is able to see the paper through Ph. 4.75, though not through Ph. 5. one's eye shows 0.25 too little; therefore, 0.25 must be added to the figures which one finds for other objects. The flame of the standard candle may also be used as a standard.

In order to avoid the influence of the central darkness scotoma, one ought to fix eccentrically in such measurements, or to use large objects, which is to be preferred. For many determinations, as for instance for the determination of brightness of white paper by daylight, and the like, the optometer is very appropriate, the patient gazing at the capital letters at a distance of $50cm$ for instance.

Scale of visual power. Professor Tscherning has suggested that the above-mentioned "photometric" scale be used for the determination of the visual power. As is well known, the test-types on Snellen's optometer are designed in such a manner as to fit into a square. The size of this square is fixed so that its side may be seen at an angle of 5′. Tscherning proceeds from a very large square, A, the sides of which may be seen at an angle of 5′ and at a distance of 600*m*. An eye which, at a distance of 6*m* is able to read a letter written in the said square, but no smaller letters, has a visual power of $\frac{6}{600}$ Snellen. This visual power was put by Tscherning at = 0. The size of the next square is A $\frac{1}{100 \cdot 25}$; seen at a distance of 6*m*, it corresponds to Snellen's $\frac{6}{420}$, or to Tscherning's 0.25. The size of the following square is A $\frac{1}{100 \cdot 50}$, and it corresponds to Snellen's $\frac{6}{240}$, or to Tscherning's 0.5. In that manner the following scale is obtained:

Tscherning's Scale:	0	0.25	0.50	0.75	1.00	1.25	1.50	1.75	2.00
Snellen's Scale:	$\frac{6}{600}$	$\frac{6}{420}$	$\frac{6}{300}$	$\frac{6}{240}$	$\frac{6}{190}$	$\frac{6}{142}$	$\frac{6}{107}$	$\frac{6}{80}$	$\frac{6}{60}$

Tscherning's Scale:	2	2.25	2.50	2.75	3.00	3.25	3.50	3.75	4.00	4.25
Snellen's Scale:	$\frac{6}{60}$	$\frac{6}{45}$	$\frac{6}{34}$	$\frac{6}{25}$	$\frac{6}{19}$	$\frac{6}{14}$	$\frac{6}{11}$	$\frac{6}{8}$	$\frac{6}{6}$	$\frac{6}{4.5}$

As is seen from the above, the numbers between 2 and 4 very nearly correspond to the numbers usually employed on Snellen's optometer, with the exception of a few intercalations only in places where the interval on the optometer is too great. According to specifications drawn by the authors of this essay, optometers have been constructed according to the new scale, the first four numbers however being omitted for practical reasons. For each row of types, the number is indicated after Snellen as well as after Tscherning. The optometer is designed to be used at a distance of 6*m*. If the room is not large enough, it is placed at a distance of 4.5*m*, but then, 0.25 must be subtracted from the numbers which indicate the visual power after Tscherning; the numerator in Snellen's fractions becomes 4.5 instead of 6.

Scale of the power of distinction. In order to determine whether one surface is brighter than another, the difference must be ($\gtrless$) a certain fraction of the brightness. This frac-

tion, which may be termed Bouguer's fraction,[1] is about $\frac{1}{100}$ for normal eyes and with good illumination. If the eye is not normal, or if the illumination is not sufficient, the fraction becomes greater. Prof. Tscherning designates the faculty of the eye of distinguishing differences of brightness as its power of distinction (D). He suggests the utilisation of the reciprocal value of the fraction as a standard measure for this faculty and, just as in the previous case, the use of the "photometric" scale:

Power of Distinction (D):	0	0.25	0.50	0.75	1.00	1.25	1.50	1.75	2.00
Bouguer's Fraction:	$\frac{1}{100^0}$	$\frac{1}{100^{0.25}}$	$\frac{1}{100^{0.5}}$	$\frac{1}{100^{0.75}}$	$\frac{1}{100}$	$\frac{1}{100^{1.25}}$	$\frac{1}{100^{1.5}}$	$\frac{1}{100^{1.75}}$	$\frac{1}{100^2}$
	$\frac{1}{1}$	$\frac{1}{1.8}$	$\frac{1}{3.2}$	$\frac{1}{5.6}$	$\frac{1}{10}$	$\frac{1}{18}$	$\frac{1}{32}$	$\frac{1}{56}$	$\frac{1}{100}$

Thus, D = 0 would correspond to cases where the patients are able to perceive the black on a white ground, but where they are unable to distinguish gray shades on the white ground.

By means of Masson's rotating disc one can easily produce the gray shades corresponding to this scale. However, as this disc is not very appropriate for the examination of patients, we have devised an optometer with gray types, the brightness of which varies between black and white after Tscherning's scale. In order to be as independent as possible of the visual power, the types are to be rather large (corresponding to Snellen Nr. $\frac{6}{45}$); it is expedient to let the patients read them for instance at the short distance of from 25 to 50*cm.*[2]

Illumination of the optometers. For the investigations in question, it is important that the optometers be uniformly and well illuminated and that the illumination be nearly

[1] The fraction was measured for the first time by the French physicist Bouguer in the 18th century. It is also met with under the name of Fechner's fraction.

[2] On account of the darkness scotoma, it is difficult for many patients when using the strongest Ph. glasses, to "find" the types, even though they are able to see the shadow. In such cases, orientation is facilitated by allowing the patient to trace the just discernible shadow with the point of a thin, black rod. By this means one obtains perhaps a somewhat higher value of the power of distinction (although it is never higher than that obtained with more practised patients), but on the other hand, the results are more uniform and more reliable.

constant. The illuminating-apparatus we use consists of a black board on which the optometers are suspended. To the board are fastened 2 arms carrying 4 connecting-lamps, each of 25 candles (see Fig. 1a and 1b) at a distance of 47*cm* from the middle of the board. After Tscherning's scale, the degree of brightness of the optometers illuminated in this manner is c = 8. If one employs another illumination, it is advisable to determine the degree of brightness in the manner already described.

Colored glasses. Besides the gray glasses, the Ph. spectacle box contains a series of colored glasses similarly constructed with gelatin plates and colored with anilin colors. In the list below are designated those parts of the spectrum which these glasses transmit. The absorption is considered to be complete, if the light transmitted amounts to $\frac{1}{1000}$ of the incident light.

I	II	III	IV	V	VI	VII
red	orange	yellow	green	blue	extreme red	bluish-green
$\lambda > 0.58$	$\lambda > 0.54$	$\lambda 0.48$	$0.59 > \lambda > 0.49$	$0.50 > \lambda$	$\lambda > 0.65$	$0.59 > \lambda$

VIII	IX	X
light blue	violet	purple
$0.57 > \lambda$	0.47λ	$> \lambda$

By combining these glasses with the gray ones, it is possible to modify colored light exactly like white light.

The compensatory adaptation of the eye. The sensibility of the eye varies continually. It reaches a constant height which at the same time is its maximum, only after a prolonged stay in darkness (sleep). The changes to which the eye is subject are of such a nature that it tries to compensate the effect of outer influences: if the illumination is intensified, a diminution of the sensibility of the eye sets in at once, and vice versa. If one places a Ph. glass of medium strength before the eye, one does not see anything to begin with, but the eye commences immediately to increase its sensibility and, after the lapse of some time, the objects become visible. The weaker the glass, the more rapidly does the change take place, and although the change of the eye is not perceptible when the weakest glasses are used, it exists nevertheless. It is easiest to demonstrate by means of comparing the images of the two eyes, that is to say,

by provoking double images by squinting, or with the help of a prism. For instance, Ph. 0.5 is placed before the one eye, keeping it there for a half or a whole minute, whereupon Ph. 0.5 is also placed in front of the other eye. The image of the first eye is then seen considerably more clearly than that of the other one[1]; in other words, the eye has increased its sensibility in order to compensate the effect of the glass. Helga Frandsen has demonstrated that, when the illumination is good and the variation of brightness not too great (from 2 to 3 Ph.), the eye may be able to compensate from 80 to 90% of the change in the course of a few minutes. If the change is greater, the compensatory adaptation becomes certainly greater, but it becomes smaller in proportion to the extent of the change. The manner in which the illumination is reduced is of no consequence. Instead of looking through a Ph. glass, the eye may be closed, or made to gaze at a piece of black paper. In case only a part of the visual field be black, the adaptation is also confined to that region (negative after-images).

The range of adaptation. The central darkness scotoma. It is well-known that one can measure the range of accommodation by means of the strongest concave glass with which one is able to see sharply at a distance. Tscherning measures the range of adaptation with the help of the strongest Ph. glass through which a maximally adapted eye is able to perceive the flame of a standard candle[2] at a distance of 50*cm*. An eye is considered to be maximally adapted after Ph. 10 has been worn light-proof before it for half an hour. If examined in that manner, the range of adaptation corresponds to 10 Ph. To obtain so high a figure, it is necessary to fix eccentrically in order to avoid the darkness scotoma: in the center the range of adaptation is c.7 Ph. The difference between the center

[1] A difference of color is also observed in the images of the two eyes; it is however not feasible here to enter into a detailed discussion of this phenomenon.

[2] The candle flame was chosen because it is an object of a definite brightness and actually within everybody's reach. If the above-mentioned illuminating-apparatus should be accepted into general use, it would be wise to measure the range of adaptation by means of a white spot on a black ground instead of the optometer, or, perhaps, to use the optometer itself. The normal range of adaptation measured in this manner would equal 8.

and the periphery is, according to Tscherning, due to the absorption in the yellow pigment of the macula which, thus, corresponds to about 3 Ph.[1]

With the help of the Ph. glasses one can easily determine the extent of the scotoma. It varies individually; in some persons it is very little pronounced. The more the illumination is reduced, the greater becomes the scotoma which corresponds to the diminution of the intensity of pigmentation outward towards the periphery.

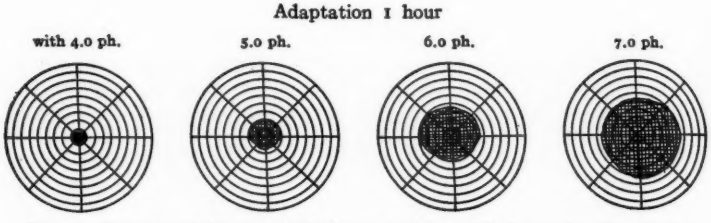


FIG. 2.—Increase of the central darkness scotoma with decreasing illumination.

The time that elapses before the sensibility of the eye reaches its maximum varies greatly and depends very much upon the illumination the eye had been accustomed to at the beginning of the adaptation. In the open air, in clear weather (sunshine), one cannot see immediately the flame through Ph. 6, and the eye must be kept in darkness for half an hour before it is able to perceive it through Ph. 10. In the evening, by lamplight, one is able to see the flame immediately through Ph. 8, the maximum being attained after the lapse of 10 minutes. If, in the latter case, the brightness is reduced by wearing Ph. 2 before the eye during a rather long time, the maximum is reached after a few minutes of complete darkness; if one has worn Ph. 5, or Ph. 6 for a longer time, one is able to see the flame immediately through Ph. 10 (see the curves Fig. 3).

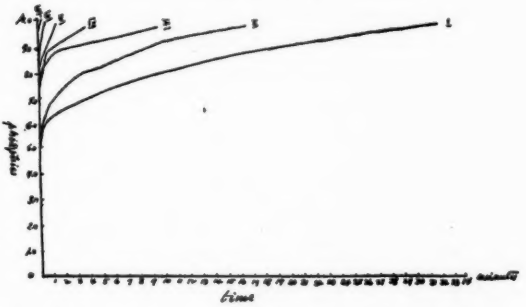
These statements refer to the peripheral sight. On account of its yellow pigment, the center is without a glass already in a

[1] The orange-colored glass, Nr. II, has been chosen so that its absorption corresponds approximately to the absorption in the pigment of Prof Tscherning's eye.

state of adaptation which corresponds to that which the periphery has, when the eye has worn Ph. 3 during some time. Therefore, the center attains its maximum adaptation (about 7 Ph.) very rapidly. When the eye goes from darkness again

FIG. 3.

Adaptation curves (see *Acta Ophthalmologica*, Vol. 1., Fasc. 4, 1923).



The photoptries are noted on the ordinate axis, the time elapsing till the light is seen being noted on the abscissa axis.

I	Curve in the open air (sunshine)
II	" " a room
III	" " " " by lamplight
IV	" after adaptation with Ph. 1,0.
V	" " " " " 2
VI	" " " " " 3
VII	" " " " " 4

over to brightness, the sensibility immediately diminishes markedly, after which it diminishes slowly, so that the time elapsing till the eye returns to its original condition is about equal to that which was necessary for reaching its maximum adaptation. A fine experiment which may be carried out by anyone is the following: blindfold one eye in the evening for about 10 minutes. If one, then, enters a dark room and removes the bandage, the difference between the vision of the two eyes is very striking. However, even though the bandage is removed five minutes before one enters the dark room, the difference is still very pronounced.

The dependence of the visual power upon the brightness. If the visual power is determined by means of a plate illuminated

by the illuminating-apparatus mentioned before (c = 8), it becomes evident that one may place Ph. 1, or even Ph. 2, before the eye without reducing the visual power. In case a stronger Ph. glass is placed before the eye, the visual power diminishes in some measure proportionately with the strength of the glass employed. If one traces the result graphically, the abscisses representing the power of the photometric glasses and the ordinates representing the numbers indicating the visual power after Tscherning, one obtains the curve (see normal curve, Fig. 4) which is composed of a horizontal part and a descending part.[1]

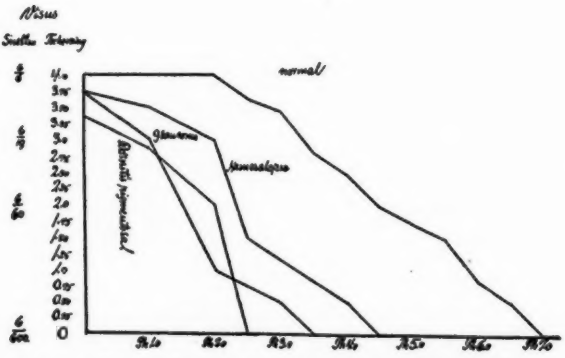


FIG. 4.—Curves showing the diminution of the visual power with a reduction of brightness, in a normal person as well as in patients suffering from hemeralopia, glaucoma, and retinitis pigmentosa, respectively. The visual power is noted after Tscherning and Snellen on the ordinate axis, the photoptries being noted on the absciss axis.

The dependence of the power of distinction upon the brightness. A similar curve is obtained by determining the power of distinction (Fig. 5 normal curve). Here, too, one may place Ph. 1, or Ph. 2 before the eye without the power of distinction being reduced.

[1] The normal curve in Fig. 4 is measured with C less than 8.
If C = 8 the horizontal part of the curve goes to Ph. 3.0 and the descending part to Ph. 8.0.

The explanation of these phenomena is to be sought for in the compensatory adaptation of the eye. To such a degree is the eye able to compensate a slight reduction of the degree of brightness, that it functions nearly as well as before. If the

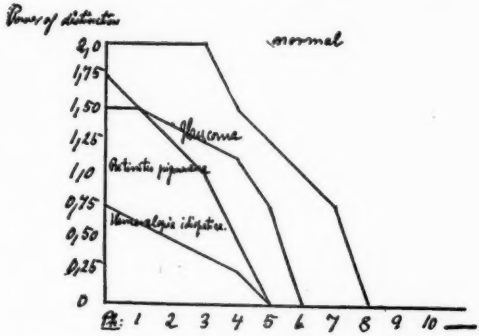
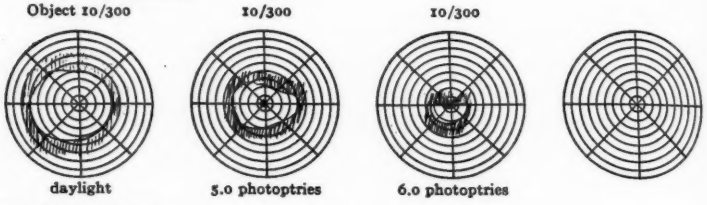


FIG. 5.—Curves showing the diminution of the power of distinction with a reduction of brightness, in a normal person as well as in patients suffering from idiopathic hemeralopia, glaucoma simplex, and retinitis pigmentosa, respectively. The power of distinction is noted on the ordinate axis and the photoptries on the absciss axis.

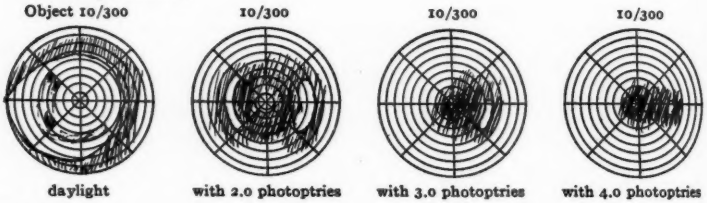
eye is not given time to adapt itself, the horizontal part of the curve disappears and the curve commences immediately to descend rapidly.

The dependence of the visual field upon the brightness. The frame in which the Ph. glasses are adjusted before the eye, covers a good deal of the periphery of the visual field. However, with a specially constructed, low frame, the visual field will be unimpaired except from 10° to 15° temporally. If a normal eye is allowed to adapt itself to a strong Ph. glass (for instance Nr. 5 and Nr. 6), and the visual field is then tested in the usual manner with a white object, no reduction of the visual field will be found. In order to examine the visual field with respect to colors, it is best to utilize colored glasses, and then to test with a white object. With Nr. VII, which transmits all the colors of the spectrum save red, no reductions in the visual field are found, even though it be combined with the strongest photometric glasses. With Nr. VI which

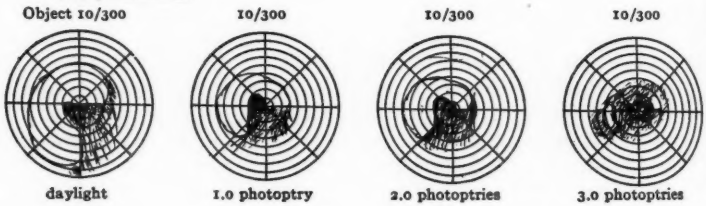
Hemeralopia idiopatica.
Visus 6/9 (Snellen).
" 3.75 (Tscherning).



Retinitis pigmentosa.
Visus 6/12 (Snellen).
" 3.50 (Tscherning).



Glaucoma.
Visus 6/9 (Snellen).
" 3.75 (Tscherning).



Amotio retinæ.
Visus 3/36 (Snellen).
" 1.75 (Tscherning).

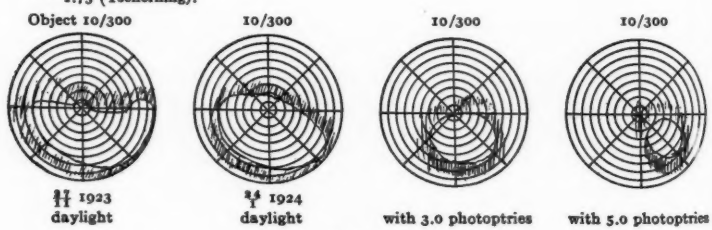


FIG. 6.

transmits the red rays only, the visual field is also found to be normal, but by combining it successively with stronger and

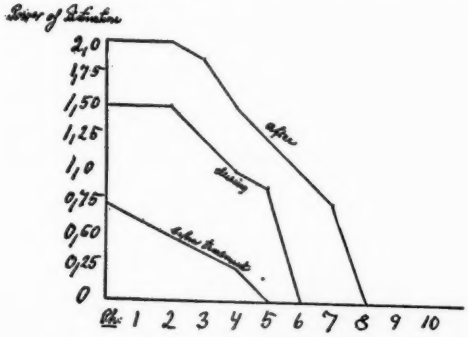
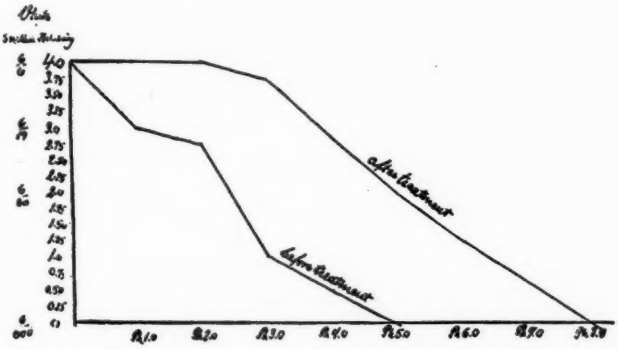


FIG. 7A and 7C.—Curves representing the power of distinction and power of vision in a patient suffering from idiopathic hemeralopia. Patient was treated with altogether 30 intramuscular injections of a preparation of fat-soluble A vitamin (produced by Professor Poulsson, Oslo, Norway—1 cm□c=/. tablespoonful of cod-liver oil. The curves were traced before the commencement of the injections, after 15 injections, and after the termination of the treatment.



stronger photometric glasses, the visual field is gradually reduced so much that, at last, with Ph. 7.75 it has an extent of only a few degrees in all directions. With Ph. glasses of

greater power than 7.75, the red color is not perceptible at all.

As to *pathological cases* we have examined patients with the following affections:

1°. Hemeralopia idiopathica.
2°. Retinitis pigmentosa.
3°. Glaucoma simplex.
4°. Amotio retinæ.

All the tests have been carried out in such a manner that the eye was blindfolded for at least half an hour, whereupon the patient was tested with the Ph. glasses. While the glasses are exchanged, the patient must keep his eye closed and cover it with his hand. With the visual field tests we used a mignon lamp as fixation object. The results of some of these tests are seen on Figs. 4 and 5 as well as on the plots of the visual field. Fig. 6.

THE TREATMENT OF TABETIC OPTIC ATROPHY.

By Dr. E. L. ZIMMERMANN, Baltimore.

(With eight field charts in the text.)

AMONG the ocular lesions of syphilis none offers so uniformly a hopeless prognosis as tabetic optic atrophy. In spite of the great advances in syphilotherapy it remains what Uhthoff (1) so aptly designated it several decades ago, "ein recht trauriges Kapitel in unserer Ophthalmotherapie."

Attempts at therapy in tabetic optic atrophy may be grouped according to drug and method of administration.

1. Prearsphenamin era: Mercury and potassium iodide.

2. Arsphenamin intravenously or intramuscularly with or without mercury and potassium iodide.

3. Introduction of drugs into the subarachnoid space or intraventricularly. (Bichloride of mercury, neoarsphenamin, arsphenamized serum of Swift-Ellis.)

1. As to the action of mercury on tabetic optic atrophy there has been considerable controversy. Alexander (2) warns against the intensive use of mercury. Long experience at Aachen convinced him that persistent treatment with mercurial inunctions not only has never improved or restored vision, but in many cases its use has actually led to rapid amaurosis.

Bing (3) states that antisyphilitic treatment in tabes is contraindicated if the optic nerve is involved. Krückmann (4) advises caution in the use of mercury. Gutmann (5) observed two patients with tabetic optic atrophy unaffected by potassium iodide who became amaurotic under mercury.

On the other hand Uhthoff (6) states that while there is no evidence that mercury ever has a beneficial action

tabetic optic atrophy, he has never observed actual injury from its exhibition.

Wilbrand and Saenger (7) differentiate two types of "spinal" optic atrophy, a differentiation which has an important bearing on therapy (mercury) and prognosis. In one type, in which functional changes indicate a diffuse involvement of the optic nerve (gradual diminution of central vision with progressive contraction of white and color fields) mercury is contraindicated. A second type consists of those cases with concentric contraction of fields with normal central vision and maintenance of color vision in the functioning portion of the visual field. Pathologically the atrophic process is limited to the peripheral portion of the nerve, while the more central portion is intact. In this type of tabetic atrophy Wilbrand and Saenger consider mercury as urgently indicated.

2. Opinions as to the efficacy of arsphenamin and neoarsphenamin have also been divergent.

Vossius (8) warns against the use of arsphenamin and neoarsphenamin as he noted rapid amaurosis follow their administration. Hensen (9) treated seven tabetic atrophies, four with neoarsphenamin and three with mercurial inunctions and potassium iodide. Neither type of treatment in any way affected the process.

A few observers have reported encouraging results. Günther's (10) patient noted failing vision in one eye in 1911, which in spite of mercury and potassium iodide progressed to amaurosis within a year. In 1916 vision in the other eye began to diminish. Examination showed a vision of $\frac{5}{10}$ and moderate concentric contraction. After an intensive combined treatment vision had improved to $\frac{5}{5}$, an improvement which was maintained at the time of the last examination, two and a half years after the commencement of treatment.

Balina (11) noted considerable improvement in three tabetic optic atrophies treated with prolonged courses of neoarsphenamin and cyanide of mercury. One result is especially noteworthy. Vision improved from $\frac{1}{60}$ to $\frac{1}{30}$ and from $\frac{2}{3}$ to 1 in the right and left eyes respectively and there was a definite increase in the size of the constricted fields.

Behr (12) recognizes three types of tabetic atrophy in which

energetic antisyphilitic treatment (arsphenamin and mercury) is contraindicated.

1. Decrease of central vision and early loss of color vision with normal or nearly normal white fields.
2. Marked contraction for white and color fields with coinciding limits for white and colors, normal or nearly normal central vision.
3. Slight perimetic changes, involving white rather than color fields, normal or nearly normal central vision with marked ophthalmoscopic changes of atrophy. Pronounced subjective visual disturbances.

Behr reports four atrophies falling into these groups treated intensively (arsphenamin, calomel, mercurial inunctions, enesol), in which a vision approaching normal had decreased to almost amaurosis in a period of one to three months. All four had originally complained of marked subjective visual disturbances.

Igersheimer (13) failed to note any influence, beneficial or injurious, of arsphenamin on the course of tabetic optic atrophy. In the type of case which conforms to the third group in Behr's classification, Igersheimer did not observe any affect resulting from antisyphilitic treatment.

3. Special procedures. Intraspinal and intraventricular therapy.

Gennerich (14) advocates intraspinal treatment (addition of neoarsphenamin to the cerebrospinal fluid of the patient) in incipient optic atrophy. Routine treatment according to him has no effect in checking a beginning optic atrophy. As soon as a peripheral contraction of the fields is noted in tabes, intraspinal treatment should be instituted. He cites a case of tabetic atrophy with contraction especially for red and green with good central vision in which a prolonged course of intraspinal treatment resulted in normal fields. No information is given as to ophthalmoscopic findings. He states that he has frequently observed such improvement in incipient tabetic atrophy as a result of intraspinal treatment.

Schacherl (15) reports encouraging results from weekly

injection of arsphenamin intravenously and intraspinal injections on alternate weeks (addition of ½ to 1 mg. neoarsphenamin to patients' cerebrospinal fluid). One patient with only central vision maintained a $\frac{6}{6}$ plus vision during an observation period of one year.

Behr (12) without giving details indicates good results with spinal drainage associated with intensive antisyphilitic treatment.

Gifford (16) observed striking improvement in six patients with "luetic optic atrophy" as a result of intracranial injections of bichloride of mercury. By his statement "we are taught to believe that so-called simple luetic atrophy is a progressive disease practically unaffected by treatment" it would seem that his report deals with the primary optic atrophy of tabes. It is to be noted that in three patients there were present central scotomas and in Case VI retinal atrophy about the papilla (spinal fluid negative!), findings not characteristic of tabetic optic atrophy. In Case I in the right eye which showed a central scotoma, visino improved from $\frac{30}{100}$ to $\frac{12}{18}$. In Case V one papilla showed advanced atrophy, the other appeared normal. It would have been a valuable addition if the type of optic neuropathies had been designated more clearly.

In analyzing the results of treatment of primary optic atrophy, it is important to determine whether the atrophy is tabetic or dependent upon cerebrospinal syphilis. Only such cases should be selected in which other symptoms of tabes are present especially where fundal or functional changes are atypical. Laboratory data (blood and spinal fluid) afford no information in differentiating between tabetic optic atrophy and a primary atrophy resulting from cerebrospinal syphilis. The following considerations are to be emphasized in diagnosis:

1. Presence of other symptoms of tabes associated with primary optic atrophy. Such primary atrophies are readily classified.
2. However it is in the monosymptomatic cases where there is only a primary optic atrophy with spinal fluid abnormalities that diagnosis becomes difficult. That optic

atrophy may occur as the isolated symptom of tabes dorsalis is a well known fact.

In Charcot's (17) classical case a primary optic atrophy developed ten years before any other evidence of the disease manifested itself. Indeed it seems probable that a "tabetic" atrophy may occur without any cord changes. Nonne's (18) case is significant in this connection. In a patient who clinically had presented a primary optic atrophy and microscopically showed changes in the optic nerve characteristic of tabes, no funicular degeneration was present.

When the primary optic atrophy does occur as an isolated symptom only a very critical consideration of functional changes and of the ultimate course place the lesion with any degree of certainty. As Wilbrand and Saenger (19) have emphasized, a neuritis peripherica in cerebrospinal syphilis may produce a descending atrophy with ophthalmoscopic and functional changes which are indistinguishable from those usual in tabetic atrophy.

From the practical standpoint of prognosis a differentiation between tabetic atrophy and that dependent upon cerebrospinal syphilis is important inasmuch as there is in the latter a greater probability of arresting the atrophy or even improving the vision markedly. The following two histories illustrate this fact.

CASE I. S. T. was first observed in September 1919 with a bilateral optic atrophy, which was diagnosed as primary in type. Examination showed aniscoria. The right eye was amaurotic. Vision in the left eye was $\frac{2}{10}$. The field for white was normal, those for red and green moderately contracted. Blood and spinal fluid were positive. There were no other evidences of neurosyphilis. An investigation of his old records showed that he had been seen at the Johns Hopkins Dispensary in March 1917 where a diagnosis of acute syphilitic meningitis with a low grade of papillitis and abducens paralysis was made. The course of the optic neuropathy supported the diagnosis of cerebrospinal syphilis. He received five courses of arsphenamin, thirty injections in all, mercurial inunctions and six Swift-Ellis treatments. The last ocular examination in April 1924, almost five years after his last visit showed a central

vision in the left eye of $\frac{1}{10}$ and no change in the color fields. Physical examination was otherwise negative. Here only a careful investigation of his past history gave an indication of the nature of the optic nerve lesion.

CASE 2. Negress, 42. Admitted to the Syphilis Clinic of the Presbyterian Eye, Ear and Throat Charity Hospital in November 1924. Complaint loss of sight.

Except for the ocular lesion, physical examination was negative. No evidence of cutaneous or visceral syphilis.

Ocular examination: Bilateral primary optic atrophy, very pale papillæ.

V. O. S. Absolute amaurosis.

O. S. $\frac{1}{10}$.

Perimetry: O. S. Moderate contraction for white, very marked contraction for red and green.

Laboratory data:

Blood Wassermann. Negative with water-bath and ice-box fixation.

Spinal fluid. 30 cells. Pandy positive.

Fixation with 0.2*cc.*

Here at first glance a diagnosis between monosymptomatic tabes dorsalis or an optic atrophy of cerebrospinal fluid seemed impossible. On questioning, a history was elicited which indicated the probability of the latter type of lesion and response to treatment confirmed the diagnosis. Eight months ago the patient had suffered severe cephalgia, especially nocturnal and in a short period diplopia developed. Under potassium iodide the headaches, though still persisting, abated and the diplopia cleared up. However six months ago she noticed vision in the right eye failing and for the past four months this eye has been completely blind. Two weeks ago the patient noticed failing vision in the left eye. As a result of ten intravenous injections of arsphenamin and the use of potassium iodide (120 grains daily) vision had improved to $\frac{1}{10}$ in the left eye and the color fields had expanded strikingly.

It is recognized that papillitis or post neuritic atrophy may occur in a tabetic, also that in exceptional cases functional changes may vary from the classical form (*i.e.*, hemianopsia, central scotomas). Fuchs (20) has recorded thirty tabetics

with central scotomas. He states that in these cases the ophthalmoscopic changes may be the same as in the usual tabetic atrophy or it may have distinctive characteristics. The pale disc may not show the usual bluish white tone, but rather a yellowish discoloration. The lamina cribrosa is not visible, the borders of the papillæ are not sharply defined and the retinal vessels are slightly constricted. In other words the cases give an impression of a low grade postneuritic process. It should be emphasized that in all these cases neuropathy was considered as tabetic only because of the presence of the other symptoms of a frank tabes. In an experience as extensive as that of Fuchs thirty cases would represent a small percentage of the total tabetic atrophies observed.

The following is a study of ten patients with primary optic atrophy, all of whom showed typical somatic evidence characteristic of tabes dorsalis. Only such patients were selected who had at least $\frac{3}{10}$ vision in one eye. Perimetric studies were made at frequent intervals, but only enough are presented in each case to show progress under treatment. In each patient the fields were taken with a white object *1cm* square and a red square of *0.5cm.*

CASE 1. R. G., 42, male, white. Referred by the late Dr. Herbert Harlan.

Infection 22 years ago. Potassium iodide, but no mercury.

In April 1922 patient by chance closed his right eye while reading and noticed clouding of vision in the left eye. A diagnosis of primary optic atrophy was made. He was given six injections of arsphenamin without any apparent effect on vision. However under an intensive course of intravenous mercury the left eye failed rapidly and the right eye became affected. For the past six months he has complained of lancinating pains, unsteadiness in walking and hesitancy in urination.

Examination showed a tabes dorsalis (primary optic atrophy, absent knee and ankle jerks, positive Romberg), also hypertension $\frac{180}{80}$. Blood Wassermann positive with ice-box fixation. Spinal fluid, pleocytosis (68), strong Pandy, fixation with *0.1cc* of fluid, a high paretic Lange Gold curve.

Ocular examination (Dr. Harlan), December 1922. Irregular pupils with anisocoria, the right pupil being oval

and larger than the left. Both Argyll-Robertson. Ophthalmoscopy. Advanced bilateral primary optic atrophy. V. O. S. $\frac{1}{100}$. O. D. $\frac{1}{10}$.

Perimetry: O. S. Contracted field for white and colors with a large sector defect involving most of the lower nasal quadrant. O. D. Moderate contraction for red and white.

Swift-Ellis treatments, twelve in all, were administered at intervals of two weeks. The patient's general condition improved and the lancinating pains practically disappeared. There was a gradual improvement in the vision of the right eye which reached $\frac{3}{10}$ at the last examination (March 1925) and the fields for white and red were as extensive as at the first examination, twenty-seven months previously.

REMARK: Apparently deleterious effect of mercury. Under Swift-Ellis therapy striking improvement in central vision maintained over an observation period of three years.

CASE 2.—E. K., 48, male, white. Referred by Dr. B. C. Catlin.

History of infection at 18. For four years lancinating pains in lower extremities. August 1920 diplopia, which cleared up in two weeks under potassium iodide. Wassermann at that time reported negative. Vision in both eyes $\frac{1}{10}$. June 1922 blurring of vision in right eye. O.S. $\frac{1}{10}$. O.D. $\frac{1}{200}$. During the next five months he received ten doses of arsphenamin (0.6 to 0.9).

Examination in December 1922, showed absent patellar and ankle jerks, positive Romberg and ulnar anæsthesia. Blood Wassermann negative; spinal fluid, 12 cells, positive Pandy, fixation with 0.1*cc* spinal fluid and a negative mastic curve.

Ocular examination: Both pupils myotic, indefinite reaction. Ophthalmoscopy under mydriasis; bilateral primary optic atrophy. Vision O.S. $\frac{1}{10}$ plus. O.D. $\frac{1}{200}$. Perimetry O.S. Fields for white and red approaching normal.

O.D. Constriction, especially above and to temporal side for white and red.

He received four Swift-Ellis treatments at intervals of two weeks without any effect on the ocular condition. The patient did not return again until August 1924, at that time complaining of urinary difficulties. The ocular condition at this time was as follows: O.S. $\frac{1}{10}$ plus, almost normal white fields, somewhat constricted red fields compared with findings in 1922. O.D. $\frac{1}{200}$, marked contraction for white and only a small crescentic patch for red on the temporal side.

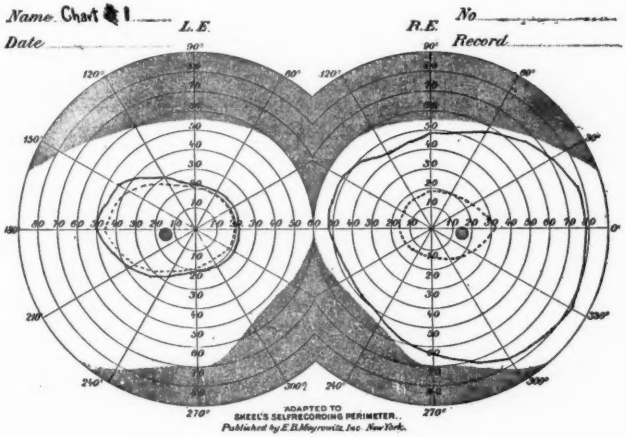
REMARK: No demonstrable effect of treatment. Though vision in left eye remained $\frac{20}{40}$ during a period of two years, the definite increased contraction of the red field indicated some progress of the atrophy.

CASE 3.—T. W., 38, colored, male, Baltimore Eye, Ear and Throat Hospital.

Frank tabes. Absent ankle and knee jerks. Positive Romberg. Paræsthesia in lower extremities. Blood Wassermann, negative. Spinal fluid, pleocytosis, positive Pandy, complete fixation with 0.1*cc* of spinal fluid, paretic Lange curve.

Ocular examination: Pupils myotic, indefinite reaction. Ophthalmoscopy, bilateral primary optic atrophy. V.O.S. $\frac{20}{30}$. O.D. $\frac{20}{25}$.

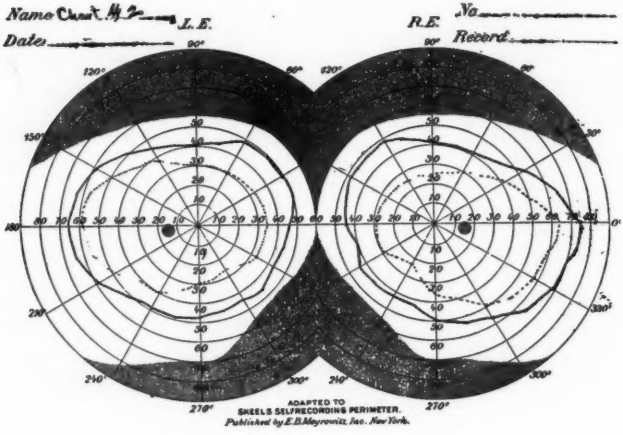
Perimetry: O.S. marked contraction for white and colors.
O.D. fairly extensive white field, marked contraction for red. (Chart 1.)



Treatment, 12 injections of intravenous arsphenamin and five Swift-Ellis treatments.

Result, O.S. $\frac{20}{30}$–3. Definite enlargement of form and color fields.

O.D. $\frac{20}{40}$ Form field, if anything slightly more contracted but red field much more extensive. (Chart 2.)



REMARK: Definite improvement in ocular function as a result of treatment. Observation period short.

CASE 4.—S.B., 44, male, white. Referred in 1921 to the Syphilis Clinic of the Baltimore Eye, Ear and Throat Charity Hospital by the Social Service Department of the Johns Hopkins Dispensary for Swift-Ellis treatments.

Diagnosis: Tabes dorsalis. Absent knee and ankle jerks, lancinating pains, girdle pains in lower thoracic region.

Blood Wassermann, negative. Spinal fluid, globulin increased, fixation with 0.2*cc* of fluid, paretic mastic curve.

At the Johns Hopkins Dispensary he had received 22 injections of arsphenamin, also mercurial inunctions and potassium iodide. For six months he had complained of clouding of vision.

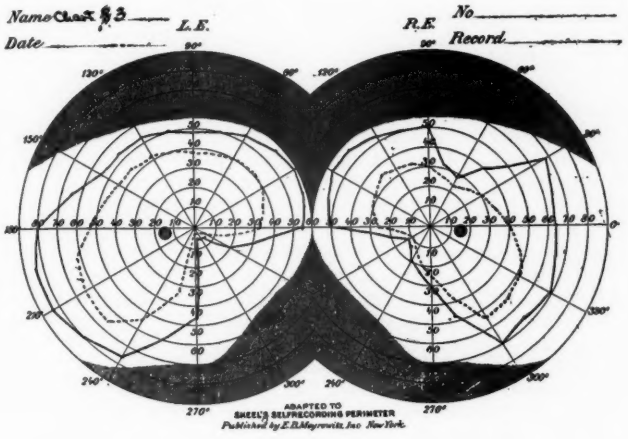
Ocular examination: March 12, 1922. Both pupils myotic, no definite reaction to light. Ophthalmoscopy, bilateral primary optic atrophy. V.O.S. $\frac{2}{10}$ plus. O.D. $\frac{1}{10}$.

Perimetry: Symmetrical fields with definite contraction, bilateral sector-form defects involving each lower nasal quadrant. (Chart 3.)

Treatment: Between March 4, 1922, and June 4, 1922, six Swift-Ellis treatments.

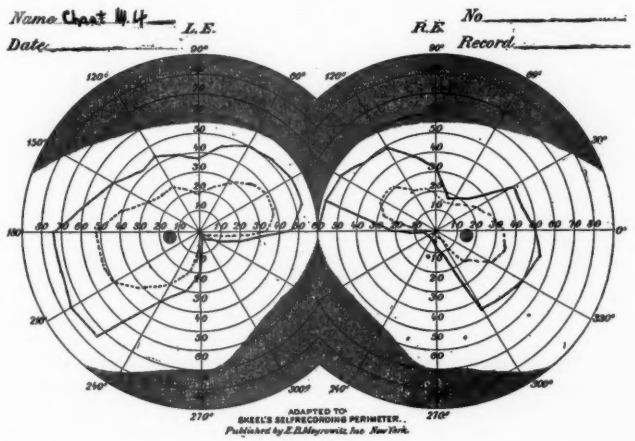
Result: Examination on June 8, 1922 showed V.O.S. $\frac{1}{10}$, O.D. $\frac{1}{15}$, fields unchanged.

In April 1924 he came to the Presbyterian Eye, Ear and



Throat Charity Hospital with the same fundal findings, V.O.S. and O.D. $\frac{3}{60}$.

Perimetry: Fields about the same as in 1922 in configuration, but more contracted. (Chart 4.)



REMARK: Treatment if anything improved central vision and had no apparent effect on peripheral vision. Unfor-

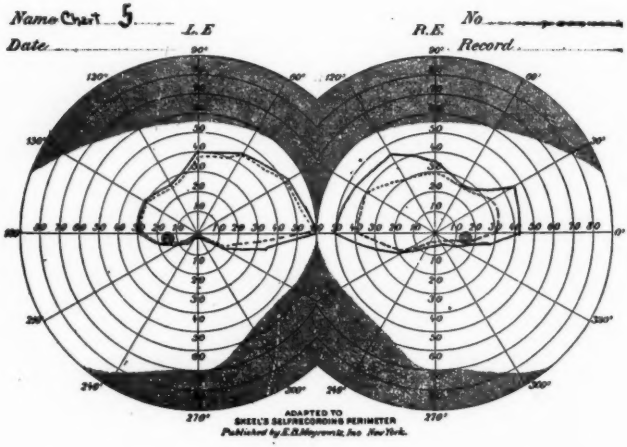
tunately the patient discontinued treatment, to return two years later with definite reduction in both central and peripheral vision.

CASE 5.—A. B., male, colored. History of genital sore at 30, local treatment only. Two years ago transient attack of diplopia. For past eighteen months numbness in lower extremities, lancinating pains, incontinence of urine at times. For past year vision has been blurred.

Physical examination: Absent knee and ankle jerks. Aortic dilatation. Hypertension $\frac{230}{150}$. Blood Wassermann positive; spinal fluid, 28 cells, positive Pandy, Wassermann positive with 0.2*cc* fluid, Lange Gold Curve 5555310000.

April 21, 1919. *Ocular examination:* Pupils equal, Argyll-Robertson. Ophthalmoscopy, advanced primary optic atrophy, bilateral. V.O.D. $\frac{20}{40}$, O.S. $\frac{20}{70}$.

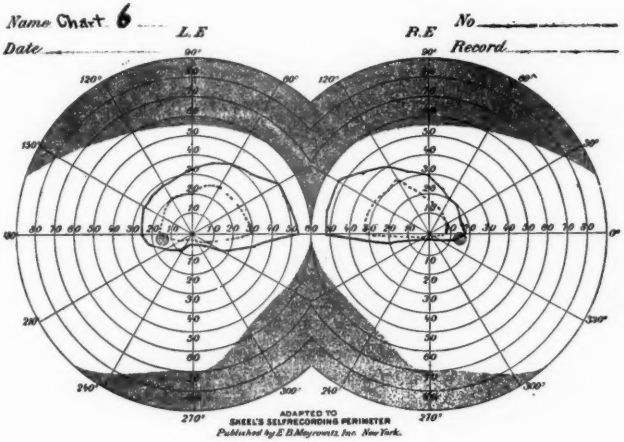
Perimetry: Fields show marked contraction for white with loss of most of lower half, red field almost coincides with white. (Chart 5.)



Treatment: Sixteen intravenous injections of arsphenamin and four Swift-Ellis treatments. Ocular examination at end of course of treatment V.O.D. $\frac{20}{60}$, O.S. $\frac{20}{60}$.

Disappeared until 1924 when he visited the Presbyterian Eye, Ear and Throat Charity Hospital stating that one month ago his vision commenced to fail and has diminished rapidly. V.O.S. $\frac{20}{100}$, O.D. $\frac{20}{200}$ with increased contraction.

Perimetry showed the same symmetrical fields as in 1919, only with increased contraction. (Chart 6.)



REMARK: In this case treatment apparently checked the process of the amaurosis. For five years vision remained satisfactory, at the end of that period to decline rapidly.

CASE 6.—C. Y., 38, male, colored. At 18 genital sore associated with suppurating inguinal adenitis, no other history. For past six months vision has been failing.

Diagnosis at Johns Hopkins Dispensary, frank tabes. Blood Wassermann, positive. Spinal fluid, 60 cells, globulin positive, fixation with 0.1*cc* of spinal fluid. Lange Gold Curve 5555553000.

Ocular examination: Sluggish pupillary reactions. Ophthalmoscopy. Bilateral primary optic atrophy. V.O.D. $\frac{20}{25}$, O.S. $\frac{20}{25}$.

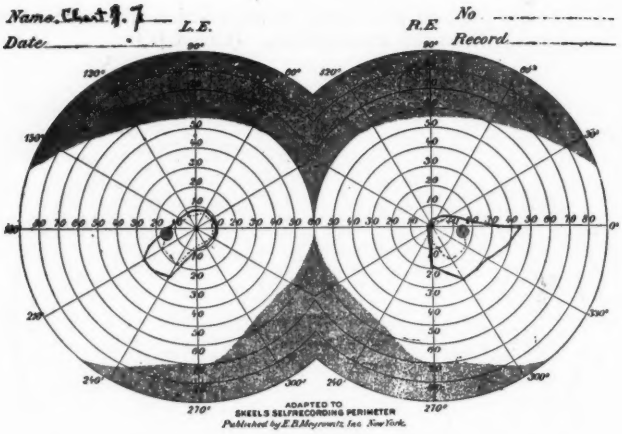
Perimetry showed a vision limited chiefly to lower temporal quadrants in each eye. (Chart 7.)

Treatment: Swift-Ellis injections on the following dates: 2/23/22, 3/17/22, 4/1/22, 4/17/22, 4/28/22 and 6/11/22.

6/19/22 V. in both eyes $\frac{20}{25}$. No color vision.

7/10/22 O.S. $\frac{20}{70}$ O.D. Hand movements.

12/29/22 O.S. $\frac{20}{70}$. Contraction for white 10 degrees, no color vision. O.D. Relative amaurosis.



REMARK: Swift-Ellis treatments apparently hastened the onset of amaurosis. During treatment loss of color vision rapidly followed by a marked reduction of central vision.

CASE 7.—C. A., female, 38, white. Baltimore Eye, Ear and Throat Charity Hospital, service of Dr. H. K. Fleck. Negative history of syphilis. For past year severe attacks of lancinating pains in lower extremities. She had difficulty in walking on streets. Port-hole vision.

Diagnosis: Tabes dorsalis. Absent ankle and knee jerks. Anæsthesia in lower extremities. Blood Wassermann, positive; spinal fluid, 40 cells, positive Pandy, Wassermann positive with 0.05*cc* of spinal fluid, paretic Lange Gold Curve.

Ocular examination: Anisocoria, both pupils Argyll-Robertson. Ophthalmoscopy: Advanced primary optic atrophy of both papillæ O.D. $\frac{3}{4}\frac{8}{8}$ O.S. $\frac{3}{4}\frac{8}{8}$.

Perimetry: Both eyes showed marked constriction for white and red, in the right within 10 degrees. The patient was given Swift-Ellis treatments on the following dates: 4/6/22, 4/23/22, 5/8/22 and 5/23/22. For twenty-four hours following each spinal injection of arsphenamized serum there occurred marked visual symptoms in the form of frequent flashes of light and vision seemed to decrease.

Ocular examination on May 3, 1922, following the second injection, showed a contraction for white within 10

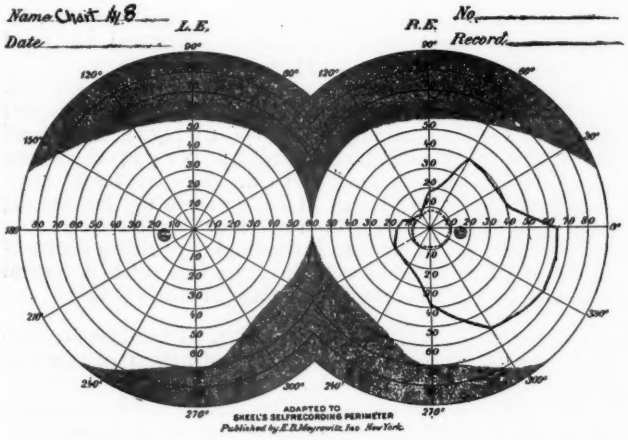
degrees in each eye with loss of color vision. V.O.S. and O.D. $\frac{15}{200}$. By the end of fourth treatment vision was reduced to hand movements.

REMARK: Following each treatment there occurred a definite Jarisch-Herxheimer reaction followed by a reduction of vision. Here the course became fulminating under Swift-Ellis therapy.

CASE 8.—C. H., 44, male, white. Referred by Dr. M. Randolf Kahn. Definite history of primary and secondary syphilis sixteen years ago. Protoidide of mercury for six months. Two years ago diplopia which lasted three weeks and cleared up under potassium iodide. One year ago patient himself noticed inequality of pupils. Vision in left eye has failed for past year.

Physical examination: Absent ankle and knee jerks, marked Romberg. Wassermann positive. Spinal puncture not performed.

Ocular examination: September 8, 1923; marked anisocoria, right pupil dilated. Both papillæ showed advanced primary optic atrophy. O.S. Light perception, O.D. $\frac{1}{2}\frac{0}{0}$ plus. Very marked contraction for white fields, with red field confined within 10 degrees. (Chart 8.)



Treatment: 24 doses of neoarsphenamin, long courses of mercurial inunctions and potassium iodide. Vision seemed unaffected by treatment, but patient is finding

increased difficulty in crossing streets, unable to see to side. October 2, 1924. O.D. $\frac{1}{10}$ plus. Vision (white and red) now limited entirely within 10 degrees. Patient disappeared for two months. Returned early in January stating that within a few days vision in right eye had rapidly failed. Examination at that time showed O.D. hand movements. O.S. light perception.

REMARK: In spite of treatment the white field was reduced to coincide with the maximally contracted red field. Following a lapse from treatment the central vision which had remained at $\frac{1}{10}$ in spite of this, diminished to recognition of hand movements during a period of one week. The effect of treatment is problematical.

CASE 9.—D. F., 39, male, colored. Service of Dr. E. A. Knorr, Presbyterian Eye, Ear and Throat Charity Hospital. Twelve years previously a genital sore followed by a rash and alopecia. At the Johns Hopkins Dispensary mercurial inunctions were prescribed over a period of six months. For the past three years he has suffered from severe attacks of lancinating pains and recently there has been some incontinence of urine. Patellar and ankle jerks absent. Blood Wassermann positive. Spinal puncture not performed inasmuch as the diagnosis was evident and no intraspinal therapy was planned.

Ocular examination: July 28, 1923. Pupils myotic, indefinite reaction. Ophthalmoscopy. Bilateral advanced primary optic atrophy. V. $\frac{1}{10}$ plus in each eye.

Perimetry: Maximum contraction for white and red. Under neoarsphenamin administered weekly (0.3 to 0.6g) the vision failed rapidly until by 10/14/23 only hand movements were recognized and by 1/10/24 there was light perception with purple vision. The treatment practically relieved the patient of his excruciating lancinating pains and resulted in a gain of eighteen pounds.

REMARK: Rapid amaurosis developing during a course of intravenous neoarsphenamin in a patient with a central vision of $\frac{1}{10}$ but with maximal contraction of fields.

CASE 10.—A. W., 33, female, white. Presbyterian Hospital, Dr. E. A. Knorr. No history of infection, one miscarriage five years previously.

Diagnosis: Tabes dorsalis. Excruciating lancinating pains in lower extremities. Absent ankle and knee jerks. Positive Romberg. Definite ataxia. Blood Wassermann, positive.

Ocular examination (Dr. E. A. Knorr): Anisocoria, Argyll-Robertson pupils.

Fundal changes of advanced primary optic atrophy, bilateral.

V.O.D. Absolute amaurosis. O.S. $\frac{3}{4}\frac{0}{0}$ with contraction of white and color fields within 10 degrees. Red and green still readily recognized.

On the basis of experience in patients 7 and 9 she was given a placebo. By October 26, 1925, vision in left eye was reduced to $\frac{3}{7}\frac{0}{0}$, by January, 1924, only hand movements, in June patient was led in stating that she had become suddenly blind two weeks previous.

REMARK: Rapidly developing amaurosis in a patient with a vision of $\frac{3}{4}\frac{0}{0}$, maximally contracted fields. No antisyphilitics administered.

DISCUSSION.

Criticism had been directed against intraspinal treatment of lesions of the optic nerve, inasmuch as experimental and theoretical considerations would indicate such a procedure as useless. However, clinical results demonstrate clearly that optic neuropathies which have not responded to routine antileutic treatment may show improvement under intraspinal therapy. Fordyce's (21) report of two cases of optic atrophy, which were studied by Knapp are instructive. The first patient had complained of recurring attacks of pain in the left eye and back of neck for several years. Both blood and spinal fluid showed fixation. In spite of intensive antisyphilitic treatment (16 injections of arsphenamin combined with mercury) vision continued to fail until left eye became amaurotic and the right eye was rapidly becoming so. As a result of intraspinal treatment vision improved in the right eye from $\frac{3}{7}\frac{0}{0}$ to $\frac{3}{5}\frac{0}{0}$ (corrected $\frac{3}{4}\frac{0}{0}$) and in the left eye from absolute amaurosis to perception of light in the outer quadrant. The second patient, infected in 1911, complained of grey spots in front of the right eye in 1918 and within three months vision was lost in this eye. He was treated for several months with intravenous arsphenamin. In spite of this he complained some months later of clouding of vision in the left eye. Ophthalmoscopic examination by Dr. Arnold Knapp showed $\frac{3}{8}\frac{0}{0}$ vision, with temporal contraction of the field. Subsequently he received twelve intravenous injections of arsphenamin, eight intraspinal treatments and thirty-five injections

of mercury. Reëxamination in March, 1920, showed vision in the left eye had been maintained at $\frac{20}{30}$.

To quote Fordyce, "It is poor judgment, to express it mildly, to treat progressing optic atrophy intravenously without definite knowledge of the spinal fluid. Records of twenty cases treated intraspinally show arrest of atrophy. Why permit such patients to progress to complete blindness when experience shows in certain cases that progress of the atrophy can be completely arrested? A patient with a progressive optic atrophy and a positive spinal fluid is not receiving proper or adequate treatment by intravenous treatment alone."

Fordyce's (21) statement, while probably too sweeping, serves to emphasize the fact that primary optic atrophy may at times respond to Swift-Ellis injections when intensive routine treatment has had no effect in checking the progress of the atrophy. In the past year a patient under my observation whose vision had progressively diminished to $\frac{20}{200}$ in each eye in spite of intensive intravenous arsphenamin and mercury has maintained his vision for approximately six months under Swift-Ellis injections. Certainly a majority of tabetic atrophies progress in spite of intraspinal treatment and in some instances the course may become fulminating under such a method of procedure.

While a group of ten cases is too small to allow any definite conclusions, yet certain important facts as to the prognosis of primary optic atrophy of tabes are suggested.

In the first place a general statement cannot be made that treatment is contraindicated in tabetic atrophy. In patient 1 there was unexpected improvement under intraspinal therapy, an improvement maintained over a period of twenty-seven months. In patient 2, under observation only a short period, central vision definitely improved and the fields became more extensive. In patients 3 and 4 there was temporary improvement as a result of Swift-Ellis treatments, which however failed to persist. After a lapse from treatment of two years they came again under observation with progressive decrease in vision. On the other hand definite injury seemed to result from treatment in patients 6 and 7 (Swift-Ellis injections) and 9 (intravenous arsphenamin).

It is reasonable to assume that the injury resulting from

arsphenamin (as in Case 7 in which for twenty-four hours after each intraspinal treatment there occurred marked subjective symptoms in the form of flashes of light followed by a decrease in central vision) is of the nature of a Jarisch-Herxheimer reaction. Behr assumes that the membrana interna limitans of the optic nerve prevents the entry of any considerable amount of arsphenamin from the vessels in the septa of the nerve; whatever of the drug does penetrate, is fixed (?) by the spirochetes in the periphery of the nerve, the minute quantity reaching the spirochetes more remote from this membrane is not enough to destroy the organisms but only serves to stimulate them and in this way produces an injurious reaction.

Jarisch-Herxheimer reactions on the part of the nervous system are common in tabes as shown by the frequency of root pains following a dose of arsphenamin. In a structure of such delicate function as the optic nerve even a moderate Jarisch-Herxheimer reactions may have unfortunate consequences. The amount of injury produced will depend upon the extent of the lesion and the intensity of the Jarisch-Herxheimer reaction. In the extent of the lesion we have a factor which can be well estimated by functional examinations (defects in central and peripheral vision). On the other hand the intensity of the Jarisch-Herxheimer reaction is variable and cannot be anticipated. This variability may be readily observed in syphilides. A roseola may show almost no change after an injection of arsphenamin or it may show a striking intensification. Accordingly it can be readily understood why an attempt to classify tabetic optic atrophies clinically with the purpose of defining in which type treatment is indicated or contraindicated, will not be uniformly successful.

The above experiences indicate that in general a tabetic atrophy with extensive peripheral fields allows a much better prognosis than one with equal central vision but marked contraction of peripheral vision.

In the first type there may be surprising clinical improvement. (Cases 1 and 2.) In no patient belonging to this group was actual injury observed from arsphenamin and Swift-Ellis injections. On the other hand the second type

in which the course is apparently rapid even without treatment (Case 10), intravenous injections (Case 9) and Swift-Ellis treatments (Cases 6 and 7) seem to have a very deleterious effect. By referring to Behr's three types of tabetic atrophy in which treatment is stated to be contraindicated, this group will be seen to conform with his second type. That such a course is not inevitable is demonstrated by Schacherl's patient with maximum contraction of fields who under intraspinal injections maintained a vision of $\frac{11}{10}$ over a period of one year.

The risk in tabetic atrophies with maximum contraction of peripheral fields should be carefully considered. Even without treatment the course is usually brief and under treatment it may become fulminating. It is essential that every patient with tabetic atrophy should be warned before the commencement of treatment that the effect of the latter may actually be a deleterious one. If it is of the type just mentioned it would seem advisable to abandon intravenous arsphenamin or intraspinal therapy. The chance of improving vision is nil and the danger of hastening the inevitable amaurosis is considerable.

With all the new and powerful drugs added to the therapy of syphilis the prognosis of genuine tabetic optic atrophy is still about as it was in the prearsphenamin era. Only one case in this group showed a really gratifying response over an extended period of observation.

The real hope in combating this unfortunate lesion lies in the efficient treatment of early syphilis and the detection of asymptomatic neurosyphilis by routine examination of the spinal fluid. Whether the introduction of arsphenamin has resulted in a decrease in the number of tabetics will only be ascertained after a long period of observation and a critical analysis of a well controlled material.

REFERENCES.

1. UHTHOFF. "Die Augenveränderungen bei den Erkrankungen des Nervensystems." *Graefe-Saemisch Handbuch der gesamten Augenheilkunde*, 11, 2A, 1911, p. 181.
2. ALEXANDER. *Syphilis und Auge*, 1889, p. 108.
3. BING. *Lehrbuch.*

4. KRÜCKMANN. *Berl. Derm. Gesellsch. Derm. Zeitschr.*, xxxiv, 1921, p. 101.
5. GUTMANN. *Berl. Derm. Gesellsch. Derm. Zeitschr.*, xxxiv, 1921, p. 107.
6. UHTHOFF. "Augenveränderungen bei Tabes dorsalis." *Graefe-Saemisch*, 11, 2 Teil, p. 183.
7. WILBRAND und SAENGER. *Neurologie des Auges*, iii, 1906, p. 520.
8. VOSSIUS. Quoted from Behr, "Zur Behandlung der tabischen Sehnervenatrophie." *Kl. Mon. f. Aug.*, 56, 1916, p. 14.
9. HENSEN. "Über Salvarsanwirkungen bei luischen Augenerkrankungen." *Kl. Mon. f. Aug.*, 63, 1919, p. 99.
10. GÜNTHER. "Therapie der Sehnervenatrophie." *Kl. Mon. f. Aug.*, 63, 1919, p. 748.
11. BALINA. "Bénéfice considérable du traitement arséno-mercuriel dans trois cas d'atrophie papillaire tabetique." *Bul. soc. franc. de derm. et de syph.*, 1922, No. 2, p. 69.
12. BEHR. "Zur Behandlung der tabischen Sehnervenatrophie." *Kl. Mon. f. Aug.*, 56, 1916, p. 1.
"Ueber die Ernährung des Sehnerven und ihre Beziehung zu der Efolglosigkeit der Therapie der tabischen Sehnervenatrophie." *Münch. med. Wochenschr.*, 1917, No. 16, p. 517.
13. IGERSHEIMER. *Syphilis und Auge*, 1918, p. 516.
14. GENNERICH. *Die syphilis des Zentralnervensystems*, 1921, p. 210, p. 233.
15. SCHACHERL. "Ergebnisse endolumbaler Salvarsanbehandlung bei tabischer Opticusatrophie." *Zeitschr. f. Nervenheilk.*, 77, H. 1–6.
16. GIFFORD. "Intracisternal Injections in the Treatment of Luetic Optic Atrophy." *Brit. J. Ophth.*, 1923, p. 506.
17. CHARCOT. Wilbrand UND Saenger, iii, 2 Hälfte, p. 519.
18. NONNE. *Syphilis und Nervensystem*, Berlin, 1921, p. 288.
19. WILBRAND und SAENGER. *Neurologie des Auges*, iii, 1906, p. 259.
20. FUCHS. "The Field of Vision in Tabetic Atrophy of the Optic Disc." *Trans. Am. Ophth. Soc.*, 1909–11, v, p. 718.
21. FORDYCE. "The Importance of Recognizing and Treating Neurosyphilis in the Early Period of Infection." *Am. Jour. Med. Sci.*, 161, 1921, p. 313.

SUBNORMAL ACCOMMODATION.[1]

By Dr. ALEXANDER DUANE, New York.

(With one illustration in the text.)

THE following paper attempts to gather together certain conclusions that I have drawn from a study of the cases of paresis and insufficiency of accommodation observed in the last twenty years. During that time it has been my practice to test the accommodation systematically and when possible repeatedly in every case of refraction examined, so that I have been able to determine in each instance whether the accommodation was normal or not. Many thousands of such measurements have been made. In not a few instances this practice has enabled me to ascertain that asthenopic symptoms, not relieved by correction with glasses, were due to accommodative weakness. My studies have further concerned themselves with the causes of different types of this accommodative insufficiency, the significance of certain associated symptoms, and the relative value of different lines of treatment. In all some 200 cases have been considered. The results are given below.

ESSENTIAL UNITY OF ACCOMMODATIVE INSUFFICIENCY AND PARALYSIS.

Weakness of accommodation may present all grades in intensity from a complete and perhaps permanent paralysis to the very slightest and transient insufficiency. There is, indeed, no sharp dividing line between paralysis and insuffi-

[1] Read before the American Ophthalmological Society, May 5, 1925.

ciency either as regards objective evidences, or subjective symptoms, or causation. It will be profitable then to group together all forms of accommodative weakness, no matter what their degree, and to consider them as a clinical whole. There is the more reason for doing this since clinically we have more often to do with the less marked and more fugitive varieties of insufficiency which are yet of considerable importance on account of the troubles they occasion.

In the broad sense in which we are here using it, then, accommodative weakness comprises both a paralysis of accommodation in which the patient cannot use his eyes for near work at all and an insufficiency in which he can use them but not without difficulty. In this regard we must remember that what determines the symptoms in any given case is not the actual amount of accommodation present but whether that amount is at or below the normal for the age. A man of forty-two with 4 D of accommodation manifest can read with perfect ease; a boy of twenty with 6 or 7 D of accommodation can hardly read at all. To those acquainted with the theory of accommodation the reason for this difference is obvious. The man has an accommodation which is normal for his age and represents a fairly large reserve; in the boy the accommodation, although absolutely larger than in the older man, is about half the normal of his age and represents no reserve at all.

Accommodative insufficiency, then, is a condition not absolute, but relative. It may be said to exist whenever the manifest accommodation is persistently below the minimum for the age of the subject as given in the table annexed.

In applying this table we must recollect that the minimum set for each age is if anything too low and that any accommodation which persistently approaches this limit must be regarded with suspicion. Such a suspicion is often justified clinically. Thus we find not infrequently, especially in persons between fifteen and twenty-five, an accommodation which being above the minimum for the age might be regarded as normal, but which is proved to be subnormal either by the fact that it is considerably less than that found in the same subject a short time before or, still more conclusively, by the fact that later it shows a considerable increase. Such an accommodation although normal for the age is not normal for the individual

and requires clinical consideration, since those who show it often have asthenopic symptoms.

TABLE OF ACCOMMODATION

AT VARIOUS AGES

In D and tenths. Near point reckoned from anterior focus of eye (14*mm* in front of cornea).

Age	Accommodation		*Maximum*	Age	Accommodation		*Maximum*
	Minimum	*Mean*			*Minimum*	*Mean*	
8	11.6	13.8	16.1	40	3.4	5.8	7.9
9	11.4	13.6	15.9	41	3	5.4	7.5
10	11.1	13.4	15.7	42	2.7	5	7.1
11	10.9	13.2	15.5	43	2.3	4.5	6.7
12	10.7	12.9	15.2	44	2.1	4	6.3
13	10.5	12.7	15	45	1.9	3.6	5.9
14	10.3	12.5	14.8	46	1.7	3.1	5.5
15	10.1	12.3	14.5	47	1.4	2.7	5
16	9.8	12	14.3	48	1.2	2.3	4.5
17	9.6	11.8	14.1	49	1.1	2.1	4
18	9.4	11.6	13.9	50	1	1.9	3.2
19	9.2	11.4	13.6	51	0.9	1.7	2.6
20	8.9	11.1	13.4	52	0.9	1.6	2.2
21	8.7	10.9	13.1	53	0.9	1.5	2.1
22	8.5	10.7	12.9	54	0.8	1.4	2
23	8.3	10.5	12.6	55	0.8	1.3	1.9
24	8	10.2	12.4	56	0.8	1.3	1.8
25	7.8	9.9	12.2	57	0.8	1.3	1.8
26	7.5	9.7	11.9	58	0.7	1.3	1.8
27	7.2	9.5	11.6	59	0.7	1.2	1.7
28	7	9.2	11.3	60	0.7	1.2	1.7
29	6.8	9	11	61	0.6	1.2	1.7
30	6.5	8.7	10.8	62	0.6	1.2	1.6
31	6.2	8.4	10.5	63	0.6	1.1	1.6
32	6	8.1	10.2	64	0.6	1.1	1.6
33	5.8	7.9	9.8	to		to	
34	5.5	7.6	9.5	72		1	
35	5.2	7.3	9.3				
36	4.9	7	9				
37	4.5	6.7	8.8				
38	4.1	6.4	8.5				
39	3.7	6.1	8.2				

BINOCULAR ACCOMMODATION

The above are the limits for each eye tested separately. For binocular accommodation the values here given for the mean limits should be increased by 1.5D for ages 8 to 15; 1.25D for ages 15 to 20: 1D for ages 20 to 30: 0.75D for ages 30 to 40: 0.5D for ages 40 to 50: and 0.3D for ages above 50.

TYPES OF ACCOMMODATIVE INSUFFICIENCY.

The following classification of cases of accommodative deficiency seems the best for clinical purposes:

1. STATIC INSUFFICIENCY. Due to undue inertia or lack of response of the lens or the ciliary muscle, although the

energy actuating the latter is normal. The failure here lies in structural deficiencies in the terminal apparatus.

2. DYNAMIC INSUFFICIENCY. Due to deficient energy on the part of the ciliary muscle. The failure here lies in the nerves and centers governing ciliary action. Comprises the following subdivisions:

A. Subnormal accommodation with pupillary involvement, *i.e.*, with either.

(a) Total iridoplegia.
(b) Reflex iridoplegia.

B. Subnormal accommodation without involvement of the pupil.

Cases of class A are due to lesions of the peripheral nervous mechanism or of the lower centers subserving accommodation (nerve terminals, ciliary and oculo-motor nerves, third-nerve nuclei); cases of class B to disturbances affecting the higher centers (the internuclear and supranuclear connections). Cases of class A are thus often unilateral; those of class B always bilateral.

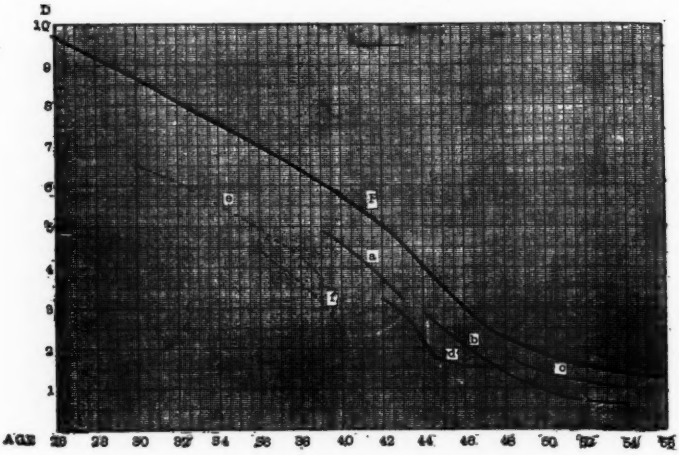
The different forms will now be considered in turn.

THE STATIC TYPE OF ACCOMMODATIVE WEAKNESS.

In static insufficiency the accommodation when examined year after year shows no irregular up and down variations but descends gradually keeping all the time close to or perchance a little below the lower limit for the age. The curve of descent, in other words, almost parallels the normal curve but keeps all the time below it—or, more strictly speaking, it represents the normal curve in a subsequent part of its course. These are really cases of premature presbyopia, in which we may say that the eye is four or five years older than the patient so that it requires assistance with reading glasses four or five years earlier than usual. Except for this such cases present few symptoms. If asthenopia is present it is usually moderate and relieved at once by the use of the proper reading glasses.

Cases cannot be positively assigned to this category unless they have been observed over a series of years, but it's pretty good presumptive evidence that they belong here if in the pres-

ence of a persistently subnormal accommodation there is a lack of subjective symptoms and particularly if there is ability to read without discomfort either with the distance correction or, if the accommodation has fallen below 4 D,



STATIC ACCOMMODATION (PREMATURE PRESBYOPIA)

P, curve representing mean range of accommodation at all ages from 26 to 56. The curves *a*, *b*, *c*, *d*, represent ranges found as a result of more or less continuous observation in cases, which from the lack of symptoms and the regular character of the descent in accommodation from year to year were regarded as instances of static accommodation. The accommodation curve in these cases almost parallels the normal curve but is considerably below it, so that the persons represented by curves *a*, *b*, *c*, *d*, have eyes which act as if they were several years older than they are, but otherwise behave normally (premature presbyopia). The curves *e* and *f* represent cases in which observations were made only at the beginning and end of the period plotted, but which from the slightness of the symptoms and the fact that at the end of the observation period the accommodation had dropped as it should if the eye were older but otherwise normal, were also judged to be cases of static insufficiency.

with a slight convex addition. It may be taken as confirmatory evidence if the accommodation though low shows no abrupt or large variations. Patients of this sort are to be contrasted with those having a dynamic insufficiency, in whom the accommodation tested at short intervals varies

widely and who read with difficulty and discomfort even when provided with what seems an appropriate addition to the distance glass.

The most reasonable explanation for cases of this type 1 is that they represent a veritable premature presbyopia—*i.e.*, an early sclerosis of the lens, the power of the ciliary muscle itself being unaffected. Thus in case *a* depicted in the figure the accommodation at thirty-nine was subnormal presumably because the lens was as rigid as that of a man of forty-two, and when the age of forty-three was reached the lens was like that of a man of nearly forty-six. Such a patient would behave in all respects like a man three years older, and the only difficulty that he would experience would be in requiring a reading glass three years before the usual time.

It might be argued that the same effect would be produced if the ciliary muscle were undeveloped but had a normal nerve supply. This seems a much less probable hypothesis than the other.

Static insufficiency is almost always bilateral, but may be unequal in the two eyes so that the necessity for a reading addition develops in one sooner than in the other. Thus in the following case—

> Major W. H. W. takes R. −0.25 −2.00 cyl. 110°; L. −0.62 cyl. 80°. At 42, accommodation R. 3.25 D, L. 3.8 D. At 43, accommodation R. 2.4 D., L. 3.2 D. and requires for reading an addition of R +1.00, L. either +0.25 or no glass. At 44–45 years accommodation R. 1.7 D, L. 2.3 D.; at 47–48 years R. 1.5 D, L. 1.6 D. At all ages well marked convergence insufficiency.

Cases of static insufficiency are illustrated in the accompanying figure.

There is no reason why cases of static insufficiency with ordinarily no symptoms to speak of should not from time to time show a nervous deficiency, too, and thereby suffer from asthenopia. In this way mixed forms of static and dynamic insufficiency may occur.

I have seen from 12 to 15 cases which I should regard as examples of static insufficiency. There were a number of others that probably belonged here but were not under obser-

vation long enough to make the diagnosis certain, and several also that would probably fall in the category of mixed cases.

ACCOMMODATIVE INSUFFICIENCY WITH TOTAL IRIDOPLEGIA.

Insufficiency or paralysis of the accommodation with complete or nearly complete absence of reaction of the pupil to both light and convergence (total iridoplegia) constitute ophthalmoplegia interior. It comprises two forms.

1. *Form, usually transient, due to poisons, such as atropine.* Of this form, so familiar to us all, little need be said. I would simply point out one or two facts.

In the first place, the effect of cycloplegic drugs on the iris is more intensive than on the ciliary body. Long ago opthalmologists found that in order to make opthalmoscopic examinations they could use a 1:5000 solution of atropine, which dilated the pupil and abolished its reaction without sensibly affecting the accommodation. The like is true of very weak solutions of homatropine. Moreover a strong solution of homatropine, which completely paralyzes the iris, rarely abolishes all the accommodation, generally from 0.5 to 1.0 D being left. Finally, we know of a few cases in which atropine intensively applied has produced permanent mydriasis, but, as Jackson says, there is apparently no case on record in which it has produced a permanent cycloplegia.

Again, the effect of a drug on the pupil is no criterion of its effect on the accommodation. Full and rapid dilatation with complete abolition of pupillary reaction may occur in patients whose accommodation never becomes satisfactorily relaxed, and per contra the pupil may dilate to only 5 or 6*mm* and yet the accommodation be almost absolutely paralyzed.

In the third place, as we all know, the effect of atropine and homatropine on the pupil is greater than that produced by a complete third-nerve paralysis, and a pupil dilated by such paralysis will be widened still further by atropine. On the other hand, the cycloplegia produced by a third-nerve paralysis is no less great and is often greater than that produced by drugs.

Finally, a word should be said about the duration of the cycloplegia produced by atropine and homatropine. It is

generally held that the mydriasis and cycloplegia disappear together—this disappearance taking place in a week or ten days after the instillation of a one-per-cent solution of atropine and in two days after the instillation of a two-per-cent solution of homatropine. But I am convinced from a large number of observations that some residual effect on the accommodation remains for a number of days after all effect on the pupil has vanished. So regularly do I find this the case that it has become my practice not to make the post-cycloplegic examination until a full week after homatropine has been used. Otherwise I feel that I am likely to prescribe too strong a glass and especially too strong a reading glass. Even for some weeks later it is not uncommon to find a rather persistent reduction of the accommodating power. Whether this is due to an actual slight residual cycloplegia or simply to the fact that the patient having once learned to relax his accommodation does not so readily resume his maximum effort I do not know. There are facts that might indicate either supposition. But in any case we must reckon with the fact that after the use of homatropine our patients may not resume their full accommodative power for several weeks. The reduction in this case below the patient's normal may amount to 1 or 2 D.

On the other hand, I agree with Jackson that a permanent loss of accommodation is never caused by a cycloplegic. At least the many thousand of measurements that I have made have never revealed such a case. It is true that I have seen a paresis lasting 20 days after the instillation of atropine. But this occurred in a boy who was scarcely a normal subject as he had suffered a concussion injury in football. More remarkable was the following:

> Helen D., aged 23. For over three years very marked asthenopia and pain produced by near work. V. $\frac{14}{16}$ each, with +1.00 sph. Accommodation, R. 6.25−6.75 D, L. 5.15 D, later falling to R. 4.25 D, L. 4.50 D. A 1.5% solution of atropine, instilled for 3 days, revealed a correction of +1.87 D, R. and L. After 14 days accommodation 1.25 D and pupil still dilated; in 21 days, accommodation 1.2 D, and in 28 days 1.5−2.0 D, falling in the course of another three weeks to 0.75 D. In the meantime, however, had an attack of ill-defined illness marked by a chill, vertigo, and severe occipital pain. Pilocarpine had no lasting effect

on the paresis but under the influence of convergence exercises and strychnine given hypodermically the accommodation rose in some months time to 6, 7, and even 8 D. Thereafter, accommodation varying greatly, sometimes falling to only 0.5 D, sometimes rising again to 6 or 7 D. The exacerbations of paresis could generally be traced to some intercurrent condition (sinus infection, tonsillar disease, streptococcus infection of the throat, intermittent malarial fever, etc.). Associated with the accommodative paralysis were various motor anomalies with at times irregular contraction of the fields.

The fact that the symptoms persisted, although in very variable degree, for fourteen years suggested some continuing cause. One diagnostician suggested a circumscribed arachnoiditis in the subtentorial region; another thought there was a hysterical element. She received treatment for the intercurrent conditions (operations on her sinuses and tonsils) and had a cerebellar exploration with evacuation of the fluid from the lateral cisterna. This seemed to produce some effect, though not a permanent one. For her accommodative paresis I gave her bifocal glasses with reading segments of strength varying from 1.50 to 2.50 D, although there were considerable periods in which she could dispense with a reading addition altogether. For the rest she was a woman of splendid physique and a good deal of energy, who did most excellent educational work among the Kentucky mountaineers.

Here was a case in which the instillation of atropine was apparently the direct cause of a continuing paresis of accommodation. But the history and still more the tests made before the instillation clearly showed that the paresis antedated the latter and was increasing at the time the atropine was used. Hence the fact that the accommodation four weeks after the instillation was only 1 or 2 D means very little. It might well have been no more than if no atropine had been used at all. The subsequent history bore out this belief. There were later great variations in the accommodation, evidently set up by external factors. This would hardly have been the case in a permanent paralysis produced by atropine.

In my belief this case was probably a condition of static insufficiency combined in a person of great nervous susceptibility with a varying dynamic insufficiency set up by intercurrent infections. This dynamic insufficiency produced the

exacerbations of cycloplegia. The cycloplegia produced by the atropine was of temporary duration—the actual effect of the drug probably acting not much longer than usual.

2. The second variety of ophthalmoplegia interior is *the more or less permanent form due to structural changes.* This constitutes one of the varieties of third-nerve paralysis and occurs either in conjunction with paralysis of the extraocular muscles or independently. Sometimes a paralysis of this sort alternates with paresis of the exterior muscles or it may alternate with an attack of headache. The paralysis of the ciliary body and of the iris may be complete or incomplete. It is not uncommon to find an incomplete paralysis of the accommodation with a complete total iridoplegia. The reverse condition—incomplete iridoplegia with complete cycloplegia—is certainly very rare in the case of structural anomalies. In this regard it must be remembered that the pupil even in complete iridoplegia is not as widely dilated as in atropine mydriasis, often being in fact only from 4 to 5*mm.* Its actual width will depend to some extent on the state of the dilatator. If the latter is paretic the pupil will be comparatively small. This seemed to happen in the following case.

J. E. B., aged 24. Syphilitic infection 5 years previously. R. accommodation 15 D, L. 8 D. R. pupil 3.5*mm* (varying); react; to light and convergence. L. pupil 3.5*mm*; reacts neither to light nor convergence and dilates only very moderately (to 5*mm*) when a 5-per-cent cocaine solution is instilled. Exterior muscles normal. Fields normal.

Here the slight dilatation of the pupil under cocaine as well as its comparatively small size would seem to argue that the dilatator was paretic.

This form of paralysis like third-nerve paralysis in general is due to many causes, but in the great majority of cases to syphilis in its later stages. We thus not infrequently find it as a sequel to the condition next to be described in which paresis of accommodation is combined with reflex iridoplegia. Indeed in a case of this sort we may find reflex iridoplegia in one eye and a total iridoplegia in the other.

These paralyses may be fairly stable, sometimes remaining

unchanged for years. At other times they pass, only to be succeeded, it may be, by paralyses of another nature.

The site of the lesion is sometimes in the third nerve or its terminal branches, but is more often—and in uncomplicated ophthalmoplegia interior it would appear almost invariably—in the nerve nuclei or their immediately adjacent efferent connections.

ACCOMMODATIVE INSUFFICIENCY WITH REFLEX IRIDOPLEGIA.

Accommodative insufficiency occurs quite frequently in conjunction with reflex iridoplegia (Argyll-Robertson pupil). These conditions are due predominantly to syphilis and almost invariably involve the nuclei and their afferent connections.

The pupil in these cases varies greatly in size. Sometimes there is miosis (spinal miosis), due either to spasm of the sphincter or paralysis of the dilatator. In this case the convergence reaction may be difficult to make out. At other times the pupil is of medium size or even rather widely dilated (up to 7*mm*). In the latter case the convergence reaction is usually marked, so that the contrast with the failure of the light reaction is very evident. This may be a source of confusion since a patient tested for light reaction may converge on the source of light and hence show a contraction which might be ascribed to the effect of the light when it is really due to the convergence. To avoid this error the test should be made as described by Uhthoff, *i.e.*, in a dark room with the light at which the patient is looking focused by a loupe directly on the cornea. The other eye is covered, and, the examined eye being then alternately shaded and uncovered, any reaction taking place must be due to light, since under these conditions neither accommodation nor convergence will be set into action.

These paralyses present all sorts of varieties. The light reaction may not be quite absent but only extremely slight and evanescent. Again, there may be all grades of accommodative paralysis. The latter is seldom complete. The condition is usually bilateral but may be present to an unequal degree in the two eyes or may even be strictly unilateral (so in three out of the seven or eight cases of this type occurring in the series here reported).

The condition may pass into a total iridoplegia, so that the pupil is absolutely immobile. In that event if the pupil had previously been contracted, it may become more or less dilated with the increase of the paralysis. On the other hand, a reflex iridoplegia instead of increasing may pass off; or if it remains the associated paralysis of accommodation may diminish.

Paralysis of accommodation with reflex iridoplegia is frequently associated with other paralyses which are often more or less evanescent. Or it may alternate with such paralyses, or may replace severe attacks of headache. The condition is generally a manifestation of syphilis in its later stages and hence is usually found only in adult life. But it may occur as an infantile or perhaps congenital manifestation. Following is an instance in a case which I saw through the courtesy of Dr. Colman W. Cutler.

> Florence S., aged 16. Mother has tabes. A short time before she gave birth to Florence her pupil dilated, but after Florence was born it contracted and now she has a regular spinal miosis. Florence had scarlet ever at the age of two years and a half, and ever since it has been noticed that the left pupil was dilated and the vision of that eye for reading poor. Accommodation R. 4.75 D, L. 2.50 D. Pupils R. 4 to 5*mm* diminishing to 3*mm* in convergence; L. 5 to 6*mm* diminishing slightly in convergence. No reaction to light in either eye.

These cases of accommodative weakness with reflex iridoplegia, it will be observed, bear in course, causation, and site a great resemblance to the cases with total iridoplegia just described. In fact, as first said, one variety may pass into the other or a patient may have accommodative paralysis associated with reflex iridoplegia in one eye and with total iridoplegia in the other.

The diagnosis between the two is not always easy. We may have cases in which the reactions both to light and convergence are absent, but in which nevertheless we are dealing with an Argyll-Robertson pupil. These are cases associated with a considerable convergence insufficiency and in which therefore, though the paths for the convergence reaction are intact, the reaction itself is not initiated, simply because there is no impulse to converge. This sort of functional

inaction of the pupil when the gaze is shifted from distance to near is found also when the light reaction is present, affording then the picture of an inverse Argyll-Robertson pupil. This condition, to a brief consideration of which I will return later, argues in general no actual interference with the motor mechanism of the iris.

Cases in which the light reaction is absent and there is only a slight convergence reaction offer special difficulties. They may be quite analogous to those just described, differing only in the fact that the convergence insufficiency is not extreme and hence the convergence reaction is not altogether lacking but is simply less marked than in normal cases. These are cases of true reflex iridoplegia. But, as Fuchs[1] has pointed out, cases with such pupillary symptoms may represent, not a reflex but a total iridoplegia, which is on the way to recovery and in which we get a convergence reaction but no reaction to light simply because in the process of recovery the former reaction begins to return before the latter. There is no doubt but that the same happens in the course of development of a total iridoplegia, the convergence reaction in this case not disappearing until some time after the light reaction, so that here again a reflex iridoplegia is simulated. This occurred in at least one case of the present series. Thus it will happen that in some cases—but not in all—in which a total apparently passes into a reflex iridoplegia or vice versa, we are really dealing all the time with the former condition in different stages. If the history were not decisive on this point, we should in a case of the sort incline to a diagnosis of total iridoplegia if there was no marked convergence insufficiency, while if the latter were present we might be uncertain whether the iridoplegia was total or reflex.

DYNAMIC INSUFFICIENCY WITHOUT IRIDOPLEGIA (FUNCTIONAL INSUFFICIENCY).

Insufficiency of accommodation not of the static variety and unaccompanied by pupillary anomalies is a clinical condition of considerable importance. It is comparatively fre-

[1] Cited by Marquez, *Klin. Monatsbl. f. Augenheilk.*, November–December, 1924.

quent, occurring I find in some 3 to 5 per cent of the refractive cases, and often causes a great deal of discomfort.

Of this class I have notes of about 175 cases. They offer certain features worthy of consideration.

1. *Relation to Motor Anomalies.*—It has been held that the motor evidence of an insufficient accommodation would be an esophoria in near vision—*i.e.*, a condition of convergence excess generated by the excessive effort employed in trying to make the weak accommodation act effectually. Now in the cases here collected, only 12 showed a convergence excess, whereas there were no less than 80 that showed the opposite condition, viz.: a convergence insufficiency. In 2 other cases there was a divergence excess, and in 11 cases other motor anomalies (paretic, etc.) were found, while in 62 cases the muscles were recorded as normal.

Convergence insufficiency, then, is the prevailing type of muscular anomaly present, being found in 76 per cent of the cases showing any tendency to deviation at all. In 22 cases the convergence insufficiency was very marked, in the two following, at least, amounting to a veritable convergence paralysis.

Mr. D., aged 18. Recently doing a great deal of microscopic work, in which he looked through the microscope with one eye and at the same time read the printed instructions with the other. Also did a great deal of minute drawing. This followed by severe asthenopia dolens. Atropine used. Stopped 3 weeks ago. V. $\frac{1}{1}\frac{8}{8}$ with +0.50 cyl. each. Accommodation 2.5–3.0 D each; occasionally a little more (but never over 5 D). Pupils large, equal, react slowly to light, but not to convergence. For distance esophoria 2–3∇ and with red glass homonymous diplopia, which as object is approximated continues down to 75*cm* from eyes where it is replaced by an insuperable crossed diplopia which increases pari passu with the approximation. At 27*cm* from intercentral base line, exophoria 8∇, at 16*cm* 18–24∇. Fields normal. Interior normal, but disks dusky and injected.

Under rest and proper hygienic conditions returned to normal.

Henry P., aged 16. Marked asthenopia dolens. V. $\frac{1}{1}\frac{8}{8}$ each with R+0.25 cyl., L−0.25+0.37 cyl. Accommoda-

tion 3 D. Pupils 3*mm* each; react sharply to light, not to convergence. Exophoria 1▽ for distance, 12–15▽ for near. Convergence near-point 35*cm* from base line, but when light is brought in from distance crossed diplopia begins to develop at between 55–60*cm* from eyes, and then as light is carried off continues out to 67*cm*. Muscles otherwise normal; no injection of disks.

Examination by neurologist indicates infantilism due to disturbances located in the ductless glands.

Later this boy recovered completely.

In these cases, as in a number of others in which the convergence is very subnormal, there was noted an inverse Argyll-Robertson pupil—*i.e.*, a pupil which reacts to light, but does not react to convergence. This, in these cases, indicates no real abnormality. The iris is perfectly healthy and would react to convergence, if there were any effort of convergence made. I may add that this inverse reaction occurs in cases in which the accommodation is normal but the convergence very deficient, thus furnishing additional proof that it is the convergence and not the accommodation that affords the main stimulus to the contraction occurring when one looks at a near object.

An interesting variety is that in which insufficiency of accommodation and of convergence is coupled with a *transient contraction of the field.* This combination occurs in neurasthenic cases, and the contracted field is simply one more evidence of the nervous exhaustion that gives rise to the insufficiencies themselves. I have notes of five well marked instances of this triad, of which the following is an illustration.

Dr. R. L. C., aged 32. Frequent frontal headaches; very marked and disabling asthenopia. V. $\frac{1}{1}\frac{8}{8}$ each with R+1.12+0.62 cyl., L+1.12+1.50 cyl. Wearing about the proper correction. Accommodation 7.5 D, falling later to 6 and then to 4.5–5.5 D; convergence near-point 113 to 135*mm;* exophoria for near points 26–30▽; well-marked concentric contraction of fields. Under accommodative and converging exercises, accommodation ran up to 8 and then to 8.5 D, and convergence near-point was brought to 70*mm*. Headaches and asthenopia entirely disappeared.

Another case of this type was furnished by a naval constructor, a man of 34 with very great asthenopia and aprosexia. In this case repeated careful examination at first failed to reveal any cause for the condition. Ultimately the removal of a tonsillar and dental infection cleared up the symptoms. Tonsillar infection and possibly intestinal toxæmia seemed responsible for the combination of symptoms in a third case, a young man of 21; in the fourth, a woman of 40, and in the fifth, a girl of 17, no cause was discovered.

In two cases a marked weakness of accommodation was accompanied by a contraction of the field without there being any convergence insufficiency.

Although convergence insufficiency is such a frequent accompaniment of accommodative weakness, the conditions are by no means necessarily alike in intensity. A considerable lowering of the accommodation often occurs with but slight insufficiency of convergence. The reverse also occurs but perhaps less frequently.

2. *Association with Congestion of Conjunctiva and Retina.*—In a large proportion of these cases, the note was made of continuous marked conjunctival injection, with sometimes swelling in the retrotarsus. In perhaps a still larger proportion, engorgement of the larger vessels of the disk or capillary injection of the latter was observed. The association was so frequent as to suggest a relation of cause or effect between the accommodative insufficiency and the congestive states or else suggest the likelihood that both were due to a common cause.

Whatever the relation, the conjunctival injection at least, often adds a good deal to the patient's discomfort and requires special consideration and treatment.

3. *Cases in which this Insufficiency in the Two Eyes is Unequal.*—In the great majority of these cases the insufficiency is bilateral. It may, however, differ in intensity in the two eyes and in a few cases the difference is considerable. The following is a striking instance.

J. H. P., male, aged 31. Severe pain in head and neck. Asthenopia with pains in the eyes. Photophobia. Vision $\frac{18}{18}$ with R -0.25 cyl., L $+0.25-0.75$ cyl. Accommodation R about 3 D, occasionally going up to 4.5 D; L usually

about 6 D sometimes less. Pupils 4.5*mm* each, react to light. Rotation outward of R eye somewhat subnormal. Convergence near-point remote (125–175*mm*). Ordinarily little deviation for near, but if he fixes sharply with left eye at an object a foot away, the right eye converges sharply behind the screen (spasmodic convergence excess). Received a grab front for reading of R+2.00 (afterward +1.25), L+0.50. Intestinal toxæmia and the presence of pus pockets about the teeth discovered. These conditions treated, especially latter. After this did pretty well. A year and a half later accommodation R 3.7, L 5.2 D. With +1.25 added R and +0.50 added L, accommodation, R 5.8, L 6.2 D. Without grab front, esophoria for near points 10∇; with grab front, if anything slight exophoria.

4. *Site of Disturbance.*—From the lack of any real involvement of the pupil in these conditions and from the fact that the disturbance of accommodation is almost always bilateral, we may infer that commonly the condition is dependent on an interference with the centers or tracts above the nerve nuclei or interconnecting the latter.

5. *Varieties.*—To be classed with cases of functional insufficiency of accommodation are those in which the accommodation, though normal in absolute amount, soon gives out on use, so that the print blurs. Of a different sort, although also belonging here, are the cases in which the accommodative adjustments, though normal in extent, are made sluggishly and with troublesome or even painful effort.

6. *Causes.*—It is very difficult often to fix the cause in these cases. But a survey of them indicates that in general we are dealing here with a functional condition which may be set up by almost any agent that depresses the vitality or diminishes the nerve power. In this regard the condition is essentially different from those already considered which are due to structural rigidity or toxic disturbance of the peripheral mechanism or to structural changes in the nuclei. Corresponding to this distinction is the fact that syphilis, which is such a frequent cause of nuclear paralysis, rarely produces this condition. If it does cause it, it is probably from the secondary anæmia that it occasions rather than from any toxic or degenerative effect.

In my series of cases the following appear as causes:

(*a*) Conditions causing direct eye strain. Among these may be mentioned continuous exposure to intense light; excessive work with the microscope, especially when the conditions are such as to dissociate the eyes (cf. case of Mr. B., *ante*), working by poor illumination, and probably working with improper glasses. One case may be cited.

> Joseph C. F., aged 30. Works 7 hours a day at electric welding, in which with the protection of a mask and dark glasses he is looking continually at an intensely hot and bright spot of light. Pain and asthenopia. Accommodation 1.8 D rising later to 2.5 D. Exophoria for near points 9▽, convergence near point 115*mm*. Field normal.

(*b*) Traumatism. Injuries to the head, *e.g.*, concussion injuries in football, as in one case noted, may cause it.

(*c*) Neurotic disposition. Neurotic subjects subjected to unhygienic conditions, overwork, or wrong illumination, readily develop accommodative insufficiency, which may be coupled with neurasthenic or hysterical manifestations, such as contracted and fatigue fields. Several cases of this sort were seen.

(*d*) Endocrine disorders (hypopituitarism, hyperthyroidism, etc.). These seemed effective in five of the cases at any rate.

(*e*) Anæmia, albuminuria, diabetes. Noted in at least seven cases.

(*f*) Disturbances of blood pressure. Noted in one or two cases, in one of which the connection was demonstrated by the result of treatment.

(*g*) Nasal obstruction and sinus disease. Noted in at least nine cases. The following shows the direct relation that often seems to obtain.

> Miss S. H., aged 24. Marked headaches and asthenopia with eye pain, not relieved by correction of the slight hyperopia and astigmatism. Accommodation varying; usually only 3 to 4 D, occasionally rising to 6 D. Operations on nose relieved the asthenopia and other symptoms and caused the accommodation to rise to 11 D.

(*h*) Infection from teeth and tonsils. One marked case has already been cited. Some six others were seen.

(*i*) Intestinal toxæmia, disease of the gall bladder, appendicitis. These conditions were noted in some eleven cases.

(*j*) Infectious diseases, especially influenza; also pneumonia, measles, whooping cough, typhoid fever, tuberculosis. Diphtheria, usually regarded as such a common cause, was recorded in but one of the cases in which the history was ascertained.

In very many cases it is impossible to fix on the cause, or, if one of the above mentioned conditions is present, it is often hard to say whether it is the sole or even the chief determining factor. Frequently we have to base our decision on the result of treatment more or less experimentally conducted.

7. *Course.*—The course varies exceedingly. The insufficiency may be of sudden or gradual development, be of very considerable amount or comparatively slight, and last a few weeks or many months. In this regard, of course, a good deal depends on the nature and persistence of the cause and the propriety of the treatment.

In any case the insufficiency is apt to be quite variable, showing a succession of more or less sudden ups and downs. This variability in fact is one of the most characteristic features of the condition; not infrequently, too, after an apparent cure, recurrences set in, which may or not be traceable to some new exciting causes. A peculiar example of the recurrent type was the following:

> Elizabeth W., aged 12. Bilious attacks since infancy. Nausea and vomiting until two years ago. For five years attacks, proceeded by severe headache, in which accommodation becomes paralyzed. Last attack four weeks ago. Since then accommodation has not cleared at all. Had whooping cough when a baby and mumps before the attacks. V. $\frac{15}{16}$, each eye, with +3.00+0.25 cyl. With +3.00 added, accommodation 4 D or less (less than 1 D net). Pupils 4.5*mm*, reacting sharply to light and convergence. Exterior muscles normal. Fundus normal. Three years later paralysis of accommodation had become permanent.

I cannot help regarding this as an atypical example of ophthalmoplegic migraine. The time of development in early childhood; the bilious attacks, turning ultimately into attacks of headache without nausea, which attacks were asso-

ciated with complete paralysis of the accommodation; the progressive increase by which the paralysis instead of being intermittent finally became permanent—all these point to this conclusion. In any event the case differed from others of the type now under consideration in that its permanence argues the presence of a structural lesion.

In cases in which both accommodation and convergence were insufficient, it has been noticed that sometimes as one function altered the other did also. This particularly in cases favorably affected by treatment of the accommodation and convergence. But in many cases the convergence insufficiency remained even after the accommodation had improved.

Some cases in which the accommodation is continually subnormal but sometimes becomes more depressed still, and in which active symptoms develop only when this extra depression occurs, would seem to be a mixture of static and functional insufficiency.

SYMPTOMS.

The symptoms of accommodative insufficiency besides the blurring of vision are asthenopia, headache, eye pain, with smarting and burning of the eyelids, aprosexia, intolerance of light, sometimes also vertigo and nausea. It is often hard to tell whether the symptoms are due to the weakness of accommodation itself or to the conditions that give rise to it (syphilis, anæmia, toxic states, nasal obstruction, etc.). Some of the symptoms, too, may spring from the associated conditions, particularly from the convergence insufficiency, which often occasions blurred vision, asthenopia, and diplopia, and from the conjunctival injection and swelling. The symptoms are usually well marked and sometimes exceedingly severe; in a small minority of cases they are slight or missing altogether. This is particularly the case in static insufficiency, in which the blurred vision, readily relieved by glasses, may be the only manifestation.

TREATMENT.

Treatment comprises the following measures:

1. *Correction of the refraction* including in the correction whatever additional glass may be needed for near vision.

In static insufficiency this may be the only treatment needed.

2. *Removal of any causes leading to direct eyestrain*, such as excessive or deficient illumination, excessive employment of the eyes on minute work or work held too close to the eyes, etc.

3. Placing the patient under conditions best calculated to *increase his resisting power.* He should have good air, proper food, adequate rest, sufficient but not excessive exercise especially in the open air.

4. *Removal of the cause* of the insufficiency if it is possible to ascertain it. In insufficiencies with pupillary anomalies, antiluetic treatment is particularly indicated and often efficacious; in functional insufficiency a searching examination for the possible causes, beginning with the most obvious, or the most probable. We look for nasal obstruction, sinus disease, infection springing from the teeth or tonsils, or disorders of the gastro-intestinal tract, and correct the faulty conditions found. We also look for and treat any anæmia or disturbed vascular relations which may occur either primarily or as the result of the conditions mentioned; and we apply the proper treatment for any endocrine disorders.

5. *Treatment of the associated conditions* like conjunctival congestion and muscular anomalies. The latter may, though not so very often, require operation.

6. In the functional cases, especially those associated with convergence insufficiency, *direct exercise of the accommodation and convergence* often have an excellent effect. I exercise the convergence with prisms, base out, the test-object being held at the reading distance. Or as a method easier to apply and sometimes better tolerated, I exercise both accommodation and convergence with the fine-line object used for measuring the accommodation. The patient is directed to cover the left eye and then fixing the right on the line, to bring the latter steadily in until it begins to blur. The same process is repeated with the other eye and then with both eyes open. In the latter case the line is brought in not only till it blurs (exercise of the accommodation) but also till it doubles (exercise of the convergence). Each exercise consists of this triple approximation repeated four or five times, and the patient does two or three such exercises a day.

The use of pilocarpine or eserine as a means of stimulating the weak ciliary muscle has seemed to me entirely useless. I have tried it repeatedly without result. On the other hand, strychnine in hypodermic injection carried to the physiological limit has seemed of distinct advantage.

The treatment often requires patience and tact, and a coördination of effort between the ophthalmologist, the patient's physician, and the patient himself.

EXCRESCENCES OF THE CENTRAL AREA OF DESCEMET'S MEMBRANE.[1]

By Dr. DANIEL B. KIRBY, New York.

(*With five illustrations on Text-Plate XIX.*)

VARIOUS abnormal conditions of the corneal endothelium have been described since the advent of the corneal microscope. There is now recognized an entity consisting of discrete excrescences of the central and peri-central portions of Descemet's membrane causing distortion of the endothelial face of the cornea. The term, "Dystrophia Endothelialis Corneæ" has been suggested as appropriate. We wish to report eight cases from private practice with Dr. John M. Wheeler.

This condition with its optical properties has been described fully by Graves (1) of London. To him is due our knowledge of the precise technique by which we are not led astray by either features of the corneal epithelium or stroma which cast shadows on the endothelial face or by pigment or other deposits or glints on, or edema of, the endothelium. It was recognized and described in 1921 by Vogt (2).

The nature of these excrescences may be understood if we consider the similarity of the three glass-like homogeneous *membranes* of the human eye.

1. The *lamina vitrea* secreted chiefly by the pigment epithelium of the retina.
2. The *lens capsule* laid down by the capsular epithelium.
3. *Descemet's membrane* laid down by the corneal endothelium. Fig. 1.

[1] Presented before the Section of Ophthalmology, New York Academy of Medicine, May 18, 1925.

ILLUSTRATING DR. DANIEL B. KIRBY'S ARTICLE ON "EXCRESCENCES OF THE CENTRAL AREA OF DESCEMET'S MEMBRANE."

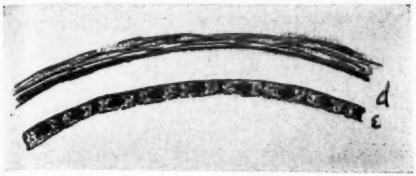


FIG. 1. Normal Descemet's membrane central area. d, Descemet's membrane. e, endothelium.

FIG. 2. Henle's warts (after Salzmann). d, Descemet's membrane. e, endothelium. w, wart.

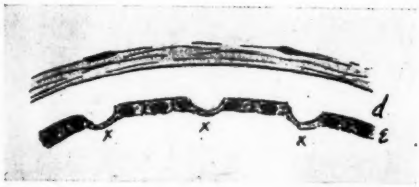


FIG. 3. Schematic representation of excrescences of the central area of Descemet's membrane. d, Descemet's membrane. e, endothelium. x, excrescences.

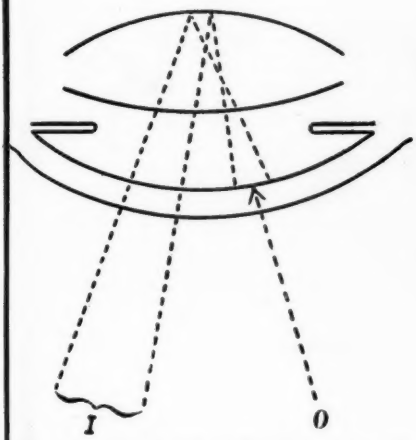


FIG. 5. Method of Retroillumination by utilizing the beam of specular reflection from the posterior lens capsule.

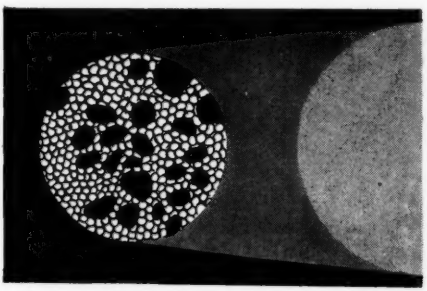


FIG. 4. Dot beam through cornea. Irregular non-reflecting areas on the endothelial face of the cornea. Seen by direct illumination with observation in the axis of specular reflection.

Now, in certain eyes, due to an abnormal stimulus, probably degenerative or protective, there occur *nodular homogeneous proliferations* of these membranes.

1. *Drusen or verrucæ* of the lamina vitrea are familiar, clinically and histologically (3).
2. *Elschnig's spheres* (4) seen after trauma to the anterior lens capsule, I regard as homogeneous non-cystic proliferations of lens capsule glass membrane by the capsular cells.
3. *Proliferations of Descemet's Membrane* (5) in the form of *Henle's warts* (Fig. 2) are well known. The excrescences of Descemet's membrane with which we are now concerned are probably similar to Henle's warts but have this feature, that they are bilateral, usually more in one eye, and start centrally and thus are of interest to the patient, and to the oculist, as by their irregular refraction and reflection, they interfere with clarity of vision. Fig. 3.

We record *five marked cases* of this condition which furnish striking pictures and *three early cases*. The ages of the patients ranged from 35 to 73 years The etiology was not discovered. There may be a familial or hereditary factor. Corrected by lenses, the *vision* in one marked case was $\frac{5}{6}$, in two others $\frac{5}{18}$, in two more $\frac{5}{36}$ and in the three early cases was normal.

The condition was demonstrated:

1. With the *ophthalmoscope* with plus 20 lens, the marked cases giving a punctate or stippled appearance, localized with difficulty on the posterior surface of the cornea. It is difficult to appreciate the finest or early cases with the ophthalmoscope.
2. With the *corneal microscope*, using the slit-beam for its detection and the dot beam for best appreciation. The details would admit of lengthy description both of features observed and also of the methods used, but I may be pardoned if I only enumerate them. The terminology is after Graves in his article in the *British Journal of Ophthalmology*, November, 1924. Both illumination and observation must be in critical focus.

a By *Direct Illumination* we observe many golden glints on the endothelial face of the cornea, due to minute specular reflections from some part of the concave mirror of the interior of each excrescence.

b. By *Direct Illumination with Observation in the Axis of Specular Reflection*, we find black or empty spaces in the endothelial mosaic, while the endothelial cells between them shine with a normal specular lustre. Fig. 4. By shifting the illumination, a glint will appear in each black space.

c. By *Retro-Illumination* from the iris a punctate or crinkled appearance is obtained. We also differentiate by this method the elements from pigment granules on the endothelium.

d. By *Marginal Retro-Illumination* we demonstrate the reversal of illumination of the excrescences showing light to dark and dark to light. The bright part of the excrescence is always on the opposite side to the reflected light.

e. By *Direct Illumination with Observation in the Axis of Specular Reflection from the Posterior Lens Capsule*, we are able to show alteration in relative illumination of the elements, depending on the depth of the focus of the microscope. The field of observation remains unaltered, while the excrescences appear darker than the field when the focus of the microscope is forward toward the patient and lighter than the field when backward from the patient. Reversal of Illumination may also be demonstrated. Fig. 5.

Most of these phenomena may be demonstrated by the above methods of illumination and observation applied to rain-drops on a window pane or better still to droplets of glycerine placed on the concave side of watch crystals or toric lenses. There is optical continuity of the excrescence or the droplet with the concave face of the cornea or glass.

CONCLUSION.—There exist in certain eyes excrescences of Descemet's membrane in the central portions of the cornea. By their irregular refraction and reflection they interfere with

acuity of vision. More of these cases will be recognized as we become familiar with the use of the corneal microscope.

REFERENCES.

1. GRAVES. *Brit. Jour. Ophth.*, November, 1924.
2. VOGT. *Arch. f. Ophth.*, 1921, Band 106, p. 102.
3. SALZMANN-BROWN. *Anatomy and Histology*, 1912, p. 59.
4. VOGT. *Atlas-Slit Lamp Microscopy.*
5. SALZMANN-BROWN, p. 37.

DACRYOPS.

A Report of Three Cases.

By Dr. OTTO LANDMAN, Toledo, O.

(With two illustrations on Text-Plate XX.)

THE first description of a lacrimal gland cyst was by Spry in 1755. Then in 1803 Schmidt described the gross appearance in two cases of Dacryops. Zur Nedden (4). The first treatment consisted of incision, or tying off a small part, of the cyst's wall, which was allowed to slough off; or a small portion of the cyst's wall was snipped off. This was the usual procedure for a long time, consequently incomplete examinations only were made, and there are no microscopical anatomical reports of dacryops, which belong to that period. Between the years of 1755 and 1905 microscopical examinations of *gland and cyst* had been made in five cases only.

CAUSES.

Dacryops may be due to congenital glandular defects, for cases have been reported in children between the ages of three and a half months up to fourteen years of age. Dor (12).

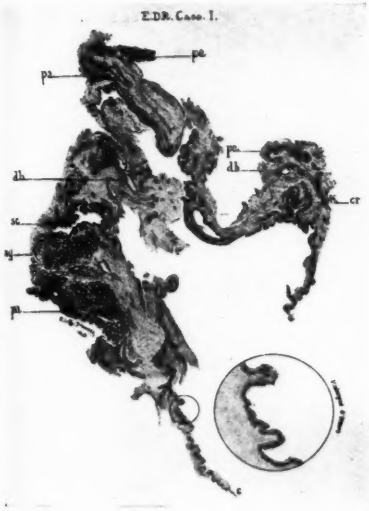
Occlusion of the mouths of the excretory ducts of the glands by accidental trauma, by chemicals, or by operations upon the tarsus for trachoma, are listed as causes.

On the other hand, intentional experimental occlusion of the excretory ducts leads not to cyst formation, but on the contrary, to atrophy of the gland. Natanson (5). Seydewitz (7).

A review of the reports on Dacryops indicates trauma as the most common cause.

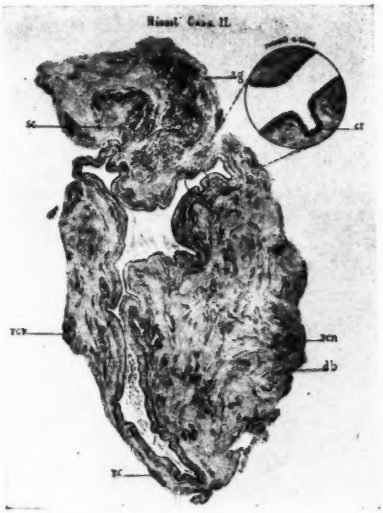
Goldzieher (8) reported his case as a retention mucous cyst

ILLUSTRATING DR. OTTO LANDMAN'S ARTICLE ON "DACRYOPS, A REPORT OF THREE CASES."



E. D. R., CASE I.

Transverse section. cc', ends of the ruptured primary cyst. pa, pigment areas of hemosiderin. db, dilated blood vessel. sc, small cyst. ag, atrophied gland lobules. cr, crypt.



CARL NIESET, CASE II.

Transverse section. Primary cyst with fibrinous contents. ag, atrophied gland lobules. cr, crypt. sc, small cyst. rcn, round cell necrosis. db, dilated blood vessel. rc, round cell infiltration. Note large areas of connective tissue and fibrous tissue with meager gland lobules.

and found goblet cells in the cells of the tubules. Piccaluga (10) is of the opinion, that although dacryops represents a retention cyst, yet it is not a mucous cyst, because by the elective methods for mucin staining, he ruled out a mucous degeneration of the glandular epithelia. In the three cases, which are about to be described, were seen the sequelæ of chronic and acute inflammation.

Mrs. E. D. R., age 56. In 1904 the patient had noticed a swelling in the outer part of the upper lid of the left eye. Stat. præsens—March 3rd, 1906. Left eye: on everting the upper lid a bluish tumor came into view. Diagnosis:—Dacryops of the orbital gland. Operation: by a curved incision over the outer third of the supraorbital ridge and cutting through the fascia, I came upon the gland and cyst which were removed. The cyst collapsed.

Microscopical Examination.—Cross section. Specimen was cut into serial sections. Hæmatoxylin and eosin.

The wall of the cyst was fibrous and lined with a double row of cells, the inner layer of cells had retained their cuboidal form and the outer layer of cells were long oval, with their nuclei mostly horizontally elongated. In some parts of the cyst's wall the cytoplasm of the cells was confluent. The wall showed numerous papillæ and directly beneath some of these, were groups of round cells, with some plasma cells. In some parts of the cyst's wall there was a single layer of cells, some had round nuclei but most of them had nuclei horizontally elongated. Under a high power, what seemed in some instances to be a single layer, was resolved into two layers with some of the nuclei, of the original second layer of cells, very faintly stained. From beginning to its end, the gland was cystic and the primary cyst extended entirely through the gland, and it had many diverticula, dipping down in various directions.

Besides the main cyst there were many smaller ones scattered about. In all of these, the linings were of a double row of cells and many of the cysts contained a fibrinous material.

Most of the glandular structure was replaced by fibrous and connective tissue; the glandular structure had atrophied, some of the secretory tubules had irregularly dilated lumen and in some of these were no lumen at all: many secretory tubules showed an alteration in the character of their cells which had lost their forms, the cell's walls had disappeared, the cytoplasm had diminished in quantity and was homogeneous, their nuclei were distributed irregularly.

Besides these changes, the cells of some tubules showed cloudy swelling, there was a marked dilatation of the blood vessels whose coats were hypertrophied; scattered throughout the gland parenchyma were pigmented areas of hemosiderin; areas of cell necrosis were found in various parts of the gland.

There were *no goblet cells* but there were vacuoles in many cells of the cystic excretory ducts.

In an excretory collecting duct on its way to the conjunctiva, were stratified cylindrical epithelia. The capsule of the gland was thickened. This specimen contained the beginning of a collecting excretory duct in the conjunctiva. The mouth of the duct was lined by stratified cylindrical epithelia which continued downwards into the duct; in its course towards the gland, further down, within the duct, the number of layers of epithelia diminished. The mouth of the duct was pressed together, by a mass of connective tissue and round cells which lay alongside of the duct and accompanied it in its downward course. There was necrosis of many of the round cells.

CASE 2.—Carl Nieset, age 13. Oct. 20, 1920. Had noticed, for some months, a swelling in the outer part of the supraorbital region of the left eye. Diagnosis: Dacryops of the orbital gland. I removed the gland and cyst through the conjunctival route.

Microscopical examination.—Transverse section serially. Hematoxylin-eosine.

There was a primary cystic invasion of the entire gland; the cyst had many diverticula and likewise papillæ. The cyst's lining rested on a firm substructure, of fibrous tissue. The cyst was lined by two rows of epithelia, an inner cuboidal, an outer flattened, with horizontally elongated nuclei. Sometimes there were short stretches of a single layer of cells, and now and then, very short stretches stripped of epithelia. Scattered throughout the parenchyma were pigmented areas of hemosiderin. There were dilated blood vessels with hypertrophied walls and groups of round cells and plasma cells, also tubules with altered cells which I regarded as a secondary effect. Most of the secretory cells had lost their granules and network in the protoplasm; the cell's walls had vanished and their cytoplasm had run together and was cloudy.

No goblet cells were found but there were many vacuoles in the cells. There were large amounts of fibrous and connective tissue and a very meager amount of glandular tissue. Besides the primary cyst, there were many small

cysts and in some of these, was extensive necrosis of their cells, likewise chromotolysis.

CASE 3.—Mrs. R. L. Aged 62. Jan. 4th, 1921. Stat. præsens:—Over the left supraorbital region was an elongated tumorous swelling. Diagnosis: Dacryops of the orbital lacrimal gland. Jan. 11th, 1921. The gland with the cyst was removed through the conjunctival route. Cyst collapsed.

Microscopical examination.—Transverse serial sections. Hematoxylin and esin.

This case differed from one and two, in this respect:—It was in an *early stage* of cyst formation with only minor cell changes. In the anterior part of the gland, were found two excretory ducts, moderately dilated but distinct from them, was a cyst which was a *circumscribed hydrops* of the capsule. The cyst had a fibrous wall with some elastic tissue, only a few cellular elements were found and close to the gland it was honeycombed. It seemed to have arisen out of the capsule. In this case there was practically no destruction of the gland tissue, but there were patches of round cells and plasma cells and a conspicuous increase of lymphoid tissue and plasma cells, lying between the tubules all over the gland. In the incipient dilatations of some excretory ducts, the inner layer of epithelium was cylindrical. Moreover these ducts had been cut obliquely and there were, now and then, some very small areas of stratified cells, but in reality, a double row of cells only lined these ducts and this was demonstrated after careful study. In such ducts were many vacuoles, but *no goblet cells.* Some necrosis was found in the cells of some of the dilated excretory ducts. There were many dilated blood-vessels with hypertrophied coats, likewise blood sinus-like spaces.

Comments.—In cases one and two, large patches of pigment of hemosiderin were found. In all of these three cases, a conspicuous feature was the dilated blood-vessels, likewise, the presence of sinus-like blood spaces. In all of the specimens were large groups of round cells and some plasma cells. The presence of a large amount of fibrous and connective tissue, with a scanty amount of glandular tissue in the parenchyma, points to a chronic inflammatory process.

Goldzieher (8) and Goerlitz (14) report the presence of goblet cells. After a most careful search of the serial sections, I did not find a *single goblet cell.*

Sata (2) and Axenfeld (3) found in normal lacrimal glands,

fat in the cells of the secretory tubules and secretory ducts. The vacuoles, which were present in the cells of these pathological specimens, which I am reporting, may have arisen from the fat having been dissolved out, by the alcohol and zylol used in the technic. In case three, Mrs. R. L., the cyst seemed formed by a circumscribed *dilation of the capsule of the gland* and agrees closely with the case reported by Lawson, which was unique ("Retention Cyst of the Lacrimal Gland," *Ophthalmic Review*, 1897). Lawson said: "There was a remarkable absence of cellular elements, and it was only after repeated examinations of many sections, that any cells could be found lining the cyst wall."

The presence of many small cysts in the parenchyma of the gland, indicated that cellular changes in either the secretory tubules or excretory ducts might cause the dacryops. Weekers (17) reported a proliferation and increase of the cells of the secretory tubules, but I found neither mytotic nor amytotic proliferation. Moreover, Weekers (17) noted more than two layers of cells in the interlobular excretory ducts, but I believe that this was not real, but was due to oblique sections. I found once only, stratified epithelia in Case 1, in an excretory duct on its way to the conjunctiva. It is commonly believed, that the pressure within the cyst destroys the inner layer of epithelia and thus leaves a single layer of epithelium in the cyst. Goerlitz (14) adds another force, namely, a stretching of the cyst's wall.

According to Zimmerman (1), in normal excretory ducts, the cytoplasm of many of the inner cuboidal cells was elongated and extended between the cells of the outer layer and reached the basal membrane, producing an appearance of a single row. I can thus see, how a stretching of the cyst wall, at such points, could result in single layer, without calling into action the pressure, within the cyst. In all of these three cases, there were areas of cellular necrosis somewhere in the gland. The contents of the cysts seemed to be a fibrinous network with a few lymphocytes.

REFERENCES.

1. 1898. ZIMMERMAN. *Archiv für Microskopische Anatomie*, Vol. 52 No. 3.

2. 1900. SATA. *Zeigler's Beitrage zur Pathologic. Anatomie*, Bd. 27, p. 570.
3. 1900. AXENFELD. *Sitzungen der ophthalmologischen Gesellschaft zu Heidelberg*. Sept., 1900.
4. 1903. ZUR NEDDEN. *Klin. M. f. A.* 1903. Bd. i., p. 381, report of literature from 1803 to 1903.
5. 1904. NATANSON. *Klin. Monatsblätter f. A.*, xli., p. 541.
6. 1904. AHLSTROM. *Klin. M. f. A.*, Bd. xlii., p. 70.
7. 1905. SEYDEWITZ, v. G. *Archiv. f. O.*, Bd. lxii., p. 73.
8. 1905. GOLDZIEHER. *Archiv. f. O.*, Bd. xli., p. 339.
9. 1906. GILBERT. *Archiv f. Augenheilkunde*, Bd. lv., p. 13.
10. 1906. CAPOLONGO. *Archiv. di Ottalm.*, vol. xii., p. 451.
11. 1906. CONTINO. *La Clinica oculist.*, p. 2509.
12. 1907. DOR. *Revue gén. d'opht.*, p. 337.
13. 1907. ORLANDINI. *Annali di Ottalm.*, vol. xxxvi., p. 585.
14. 1908. GOERLITZ. *K. M. f. A.*, vol. xlvi., p. 406.
15. 1909. REUSS. *Ber. Klin. Monatsbl. f. A.*, ii., p. 124.
16. 1909. WELIKANOFF. "A case of dislocation of the lacrimal gland and dacryops." *Westn. Ophth.*, ii., 2, p. 120.
17. 1909. WEEKERS. *Archiv. d' Ophtalm.*, p. 203.
18. 1910. KIEHLE. *J. A. M. Association*, Feb. 12th, 1910.
19. 1911. THOMPSON. *The Ophthalmoscope*, p. 396.
20. 1913. LA COMPTE. *Ann. d'oculistique*, October, 1913.
21. 1914. BRIDE. *Ophthalmic Review*, April, 1914.
22. 1914. PICCALUGA. *Annali di Ottalm.*, xliii.
23. 1922. CANAVAN. "Histology of the superior lacrymal gland in mental disease and defect." *Journal of Medical Research*, Vol. xliii., No. 4, p. 447.
24. 1922. VAN DUYSE and VAN LINT. *Archiv. d'opht.*, Bd. 39, M. 6, pp. 331–339.
25. 1922. STREBEL. *Über Kleinzystische Veränderung der unteren Tränendrüse als Ursache von Tränenträufeln.*
26. 1923. BATES. *Amer. J. of O.*, Nov., p. 930.
27. 1923. "Rückfälliger eitriger. Dacryops." *Z. f. A.*, Bd. 51, p. 337.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. DAVID H. WEBSTER, Secretary.

MEETING OF MAY 18TH, 1925. DR. CONRAD BERENS, CHAIRMAN.

Dr. Daniel Kirby reported (by invitation) seven cases of an **abnormal condition of Descemet's membrane and corneal endothelium.** (Published in full.)

Discussion: Mr. B. Graves said there is very little to add to this epitome. The condition has no distinct term to describe it, and thus dystrophia endothelialis corneæ has been suggested. He had had for several years such cases under observation, one case especially complaining of seeing through ground glass. Mr. Harrison Butler upon examining this patient's daughter found the same variety of corneal condition. In his other cases he was not able to find that heredity played a part.

Dr. Johnson: It has been my good fortune to see these cases with Dr. Kirby. The more we see of these cases the more we are sure that they are proliferation of the glass membrane, but are not satisfied as to the origin. Parson doubts that a single epithelial cell could so degenerate, and become so large. He also says that the epithelial cells are single, and if they degenerated they would not be intact. In our studies we could find the epithelial cells intact. This condition seems the same as that described by Henle beginning in the periphery. Mr. Graves was the first to describe the condition in the center of the cornea, spreading to the periphery.

In demonstrating the **technic of slit-lamp examination of the lens,** Mr. Basil Graves of London said that when a beam

of light goes through the eye which is composed of tiny particles with the different indexes of reflection, the beam of light is visible. When this beam of light is sharply and intensely defined, the visibility is so definite that it creates an optical section. In order to create an optical section it is essential that the focusing of the illumination is perfect, this is at least fifty per cent of the examination. He next illustrated by diagram the disassociation areas of the lens. In order to study the anterior half of the lens one should focus on the anterior capsule and the sub-capsule line, and in examining the posterior half of the lens, the focus used is that of the posterior capsule and sub-capsular line, which focus can also be used in examining the anterior portion of the vitreous. He called our attention to the fact that when the posterior *y* was in focus, that in order to bring the anterior *y* into view it was necessary not only to adjust the microscope, but the direction of the beam of light, also that in outlining in axis mirror reflection, the vertical from the area looked at, must bisect the angle between the light beam and the microscope.

DISCUSSION: Dr. SCHOENBERG: There is nothing which I can add to what Mr. Graves has said. The points which he has so clearly brought out and explained, I, as a beginner, have stumbled upon without knowing their explanation. Some have asked why it is worth while to examine the lens with the slit-lamp. There are several reasons for this which I would like to emphasize. First, we can study the embryonic structure. Second, in myotonic dystrophia, there is a characteristic change in the lens. We must not trust to the slit-lamp exclusively. My own method is the examination first with the binocular loop, then the slit-lamp.

Dr. KRUG: I have been using the slit-lamp since my return from Switzerland last fall, and I think after all the most valuable part is the important field of details it reveals to us especially in beginning cataract.

Mr. GRAVES in closing the discussion stated there were one or two quite important points touched upon. First: To get an instrument and use it daily, and second to use the low magnification. As to the question of whether it is of academical or real value, those who use it soon find they can make a positive diagnosis, when previously they were in doubt.

Dr. HARRY A. GOALWIN read (by invitation) a paper on the **clinical value of optic canal roentgenograms.** There is, with the exception of the sella turcica, no deep structure of the skull that can be as easily demonstrated on the X-Ray picture as the optic canal. Nor is there any in which slight changes are as important in diagnosis or prognosis.

The value of an o. c. Roentgenogram is dependent on the technique used, hence the principles are given: The central ray must coincide with the axis of the canal and strike the plate at right angles. This presupposes a knowledge of the direction of the optic canal, which has apparently not been measured by any previous author. The author has made measurements on 100 skulls to determine the size, shape and direction of o. c. in skulls of various ages. He also made a mathematical analysis of the relation between actual and Roetgenographic diameters of the o. c. He plotted these directions in the form of curves with the ages as ordinates and the inclinations of the o. c. axes to the principal planes of the skull as abscissas.

Demonstration of slides:

(1) giving direction of o. c. at various ages.
(2) o. c. of 3 yr. old child.
(3) o. c. of normal adult.
(4) o. c. of adult oxycephalus.
(5) o. c. of adult scaphocephalus.

(pointing out orientation on the Roentgenogram.)

The actual diameters of the o. c. are calculated by the formula derived by the author: $x = d - b/a\,(f - d)$.

Demonstration of slide explaining formula:

A complete analysis of an o. c. Roentgenogram should consist of the following: shape of cross section, size, changes within lumen, structure of the walls, changes in the adjacent structures (sinuses, sella, etc.) Discussion of the effect of various lesions and their various locations on the elements mentioned.

The types of cases in which an o. c. Roentgenogram is indicated, follow:

1. Optic neuritis, choked disc, retrob. neuritis, o. n. atr., etc.

2. Sphenoidal disease with or without o. n. symptoms.
3. Orbital, ocular or optic nerve tumors.
4. Known or suspected fracture of the skull, recent or healed.
5. Foreign body in the orbit.
6. Skull deformities with visual disturbances.
7. Tumors of anterior or middle fossa.
8. Certain general diseases, like Paget's, acromegaly, rickets, etc.
9. Cases of tuberculosis with optic nerve symptoms.
10. Medico-legal cases.

Demonstration of o. c. Roentgenograms:

1. Enlargement of o. c. by destruction of its post. portion by tumor of sphenoid.
2. Constriction and deformity of o. c. by tumor of orbital walls.
3. Constriction of o. c. by chronic sphenoiditis.
4. Hyperostoses and exostosis of o. c. (in case with 4-Wassermann reaction).
5. Normal canal in a medico-legal case.
6. Deformed, constricted o. c. in oxycephalus med.-legal case.
7. Case of oxycephaly with alcoholic neuritis.
8. Plagiocephaly in a case of retinitis pigmentosa.
9. O. C. in a case of cyst of the hypophisis.
10. Destruction of roofs of o. canals by a sphenoidal tumor.
11. Enlargement of o. c. in a case o. n. tumor.
12. Tuberculosis of the sphenoid.
13. Chronic periosteitis (?) of the o. c. in a case of lues.
14. Fracture of the orbit extending through the o. c.

DISCUSSION: Dr. SCHOENBERG: Three years ago I took a course in Vienna on the X-Ray of the head, and now with Dr. Goalwin at the Knapp Memorial, we are able to study the X-Ray in conjunction with the clinical picture. This work throws a new light on the pathological conditions surrounding the optic canal. There are three points which I want to emphasize. First—the technic of taking a picture, second—he has projected the canal, so that it is not distorted, third—

he has measured it mathematically. **Dr. Goalwin** in ending the discussion, said I wish to emphasize the mathematical deductions which make the measurements. The following conditions are those in which it is of the greatest value: Optic neuritis, choked disc, sphenoid diseases, tumors, suspected fractures, large foreign bodies in the orbit, causing fracture, skull deformities, tumors of the anterior or middle fossa, Pagets disease, tuberculosis with optic nerve symptoms and medico-legal cases.

Dr. M. URIBE TRONCOSO read (by invitation) a paper on **a new anatomical notation for the visual field.** The author emphasized the importance of the examination of the visual field, and the need for the simplification and improvement of our present technique. One of the serious difficulties the beginner has to deal with, is the way of recording the visual field. As mapped now the findings in the perimeter need to be inverted twice. The first is the physiological inversion of the retinal inages. The second is made necessary by the faulty habit of considering the visual field in relation with the observer's eye. This habit is in complete violation of the anatomical rule which requires the subject to be always considered standing in front of the observer. In a previous paper presented to the International Congress of Ophthalmology in Washington, 1922, the author emphasized the confusion and misunderstanding that the departure from this basical anatomical rule has produced in all ophthalmological descriptions.

The present method of recording the visual field probably originated with the use of the campimeter, in which the examiner stands behind the patient. With the advent of the perimeter the observer had to place himself in front of the patient, a new position which required a readjustment of the mental picture of the visual cones of the patient, and also a second inversion of the findings in order to be able to map them in the old diagrams. This second inversion is difficult, especially in the intermediary meridians, which must be recorded at the same inclination in the opposite side; the right side of the patient being in front of the left side of the observer. The results of this intricate and confusing method are particularly evident when the observer tries to determine to which part of the field corresponds a given defect or lesion

in the retina. With the ophthalmoscope these are visualized in its proper position, but when the observer tries to refer them to his own eye a great mental strain is necessary to locate the place of the defect or scotomas. The position of the blind spot in each side, the location of a defect in the field in cases of partial thrombosis of the retinal arteries, and the location of homonymous or heteronymous hemianopsias in the visual cones of the patient, are given as examples of these needless and complicated inversions.

The author advocates the recording of the visual field in the anatomical position, that is as found in the perimeter, in front of the patient, with only the normal inversion. He also advocates the use of a uniform graduation for the meridians in the chart, and represents them exactly as the axis of the cylinders in the trial frames, with the O always to the left of the patient and 90° above. No change in our instruments is necessary, except for the campimeter which should be made of cloth, for thrusting pins, and mark the limits of the field on a graduated side facing the operator. New charts must be made not exactly inverted but as seen by translucency.

The field of fixation will also be much more easily recorded in the anatomical position. The location of the false image in the space is better understood when referred to the new situation of the image outside of the macula in the retina of the paralytic eye.

DISCUSSION: Dr. DUANE: At first this would seem to be somewhat of an academical question, but we should have a standard. It would be of great use in transferring your plotting from the tangent screen to the chart. **Dr. Lloyd:** The original idea of the perimetic observation by Foster, centered the chart about the disc. This was in vogue for a number of years. This might be added to the anatomical idea.

DISCUSSION ON "THE FUNDUS OCULI IN GENERAL MEDICINE."

THE MEDICAL SOCIETY OF LONDON (ENGLAND).

REPORTED BY MR. H. DICKINSON, LONDON.

At the meeting of this Society on March 9th, under the Presidency of Dr. Eustace M. Callender, the subject for discussion was "The Fundus Oculi in General Medicine." It was opened by:

Mr. ERNEST CLARKE, who said the service of the ophthalmoscope in medicine had been enormously increased during the last few years, especially since the introduction of the luminous ophthalmoscope, the latter advance having brought the subject of ophthalmoscopy within the practical reach of every medical practitioner. As this latest developed instrument could be brought almost into touch with the cornea in use, the darkened room was not needed, the patient could be examined in any posture, and interference of observation by refractive troubles was almost nil.

It was one thing to see the fundus, quite another to interpret what was seen. One authority stated there were 90 varieties of "normal" fundus, a number which was probably not an exaggeration if included in this was the great variety of vessel distribution. Only by observing many fundi, both normal and in disease, could the practitioner recognize departures from the usual. Failing this experience, he was apt to be alarmed when he saw gross changes, which might not be of clinical significance, such as opaque nerve fibers and colobomata, whereas he might miss the changes which the ophthalmoscope showed to be present in arterio-sclerosis.

In former years it was not easy to obtain accurate fundus

pictures for study and comparison; it was important to avoid wearying the patient, and the eye must not be illuminated too long at a time. On the suggestion of Dr. Rayner Batten, Messrs. Hamblin had now set up a studio for this work, and at a few minutes' notice they undertook to take a picture of the back of the eye while the patient was under the mydriatic; they also would take a series of pictures of the same eye, enabling the ophthalmic surgeon to compare the conditions, without having to trust either to his memory or to a hasty sketch and a few words of description. Mr. Clarke said drawings in color were always more useful than those in black and white. Lantern slides of these pictures could also be made, hence it was a great accession for teaching purposes.

Mr. Clarke showed, on the epidiascope, a large series of colored drawings to illustrate his thesis, the illustrations being arranged to illustrate the changes seen in connection with various diseases.

Dr. F. PARKES WEBER said that once when he was examining the fundi of a patient he died, and, continuing his examination, he noticed that the columns of blood in the vessels took on segmentation, i.e., the actual blood was interrupted by small spaces, and these spaces could be moved up and down by exerting pressure on the chest. Later, of course, the cornea became too cloudy and opaque for the observation to be continued.

Dr. RAYNER BATTEN agreed as to the importance of having consecutive drawings in many cases, so as to determine in what direction a case was going. It was difficult to classify fundus pictures according to the diseases, and the difficulty was increased by the reluctance of the medical man sending the case to provide notes of the patient's condition. Such a note was very important, and should in all cases be supplied.

Dr. Batten also projected a number of very instructive slides from his series.

Dr. JAMES COLLIER, a neurologist, said that an ophthalmoscopic examination should be made in every case of illness whatsoever. It was a superficial examination which could be done in a moment, like looking at the tongue or feeling the pulse, and it afforded valuable information. The beginner could and should practise on normal subjects; only in that

way could he know when he was in the presence of the abnormal. Every student in his wards was obliged to use the ophthalmoscope on every patient; he reported what he saw, and the speaker criticized his report or confirmed it. He said American physicians had remarked to him that in England more attention was given to the ophthalmoscope than in America. In many diseases the first symptom might be ocular; but even more important than these positive findings were negative reports that the fundi were normal. Recurrent headaches, unexplained vomiting, transient blindness, giddiness, and many other symptoms suggested a gross intracranial lesion, and when there was no ophthalmoscopic examination it was impossible to treat the case without having a lurking suspicion that there was a serious organic underlying condition, and that therefore valuable time was being wasted. He had seen difficult diagnostic problems settled by recourse to the ophthalmoscope. Of this he related a number of instances.

Sir WILLIAM WILLCOX agreed that if the ophthalmoscope were used on every case, much more knowledge would be available on the relationship of changes in the eye to general diseases, and diagnoses would be more accurate. He had recently been much interested in the changes in the fundus oculi from toxæmia. Until comparatively recently changes in the fundus were considered to be due to one of three conditions: diabetes, Bright's disease, high blood pressure. But it was very common to find marked changes such as hæmorrhages in the fundus in cases having no increased blood pressure and in which the renal function was not disturbed. In such cases he considered it most important to search for possible channels of infection: teeth, antra, sinuses and so on. He had seen a number of toxic cases with retinal changes, and he removed the focus, following it by Plombière treatment, to eradicate secondary intestinal invasion, and then the retinal condition cleared up. Certain drugs appeared to have an affinity for retinal tissue, and among these were atoxyl and quinine. And veronal seemed to have a lasting effect; he was still seeing cases showing a curious nervous effect of the administration of that drug during the war. He suggested that persons having retinitis showed themselves to be sensitized to

the toxin, and when giving a vaccine to such a commencement should be made with very small doses, a few thousands, instead of the millions usually given. He regarded this sensitization as akin to that in asthma and angio-neurotic œdema.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M. D., F. A. C. S., New Rochelle, N. Y.

(*Continued from July issue.*)

IV.—ORBITS AND ACCESSORY SINUSES.

81. Aniceto-Solores. **Hydatid cysts of the orbit.** *Archiv d'Ophtalmologie*, 1921, Nos. 7 and 8, pp. 406 and 491.

82. Becker, H. **Congenital microphthalmos of both eyes, with exophthalmos and carcinoma in the left orbit.** *Deutsche Ophthalm. Gesellschaft*, Jena, June, 1922.

83. Bedell, A. J. **Osteoma of the orbit.** *American Journal of Ophthalmology*, February, 1924.

84. Begle, H. L. **Hæmangioma cavernosum of orbit.** *Ibid.*, November, 1923.

85. Benedict, W. L. **Tumors and cysts arising near the apex of the orbit.** *Ibid.*, March, 1923.

86. Cange. **L'osteomyelite aigue des petitesailes du sphenoide.** *Archiv d'Ophtalmologie*, 1921, p. 321.

87. Cange. **Les mucoceles geantes du sinus frontal a evolution orbitaire.** *Ibid.*, 1922, xxxix., No. 4.

88. Doherty, W. B. **Formalized cartilage implants following enucleation.** *American Journal of Ophthalmology*, January, 1923.

89. Hoeve, V. D. **The optic nerve and the accessory sinuses.** *Klin. Monatsbl. f. Augenheilkunde*, lxviii., p. 691.

90. Lapersonne, Veltor and Prelat. **Deux surnumeraires developpees dans l'orbita.** *Archiv d'Ophtalmologie*, 1921, p. 129.

91. Latteri. **Osteoma del Frontale con sviluppo endorbitario.** *Annali di ottalmologia e clinica oculistica*, 1922, Nos. 3–5.

92. Lunding-Smidt and Jensen. **Un cas d'encephalocele posterieure de l'orbite.** *Archiv d'Ophtalmologie*, 1922, xxxix., No. 2.

93. Marzio, di. **Cloroma dell'orbita.** *Annali di ottalmologia e clinica oculistica*, 1922, Nos. 6–9.

94. MEISSNER. **Tubercle bacilli in the orbital tissues.** *Zeitschrift f. Augenheilkunde*, xlvii., p. 101.

95. MORGAN, J. A. **Gunshot wound of orbit, operation, recovery.** *American Journal of Ophthalmology*, February, 1923.

96. PFINGST, A. O. **Knife blade in the orbit.** *Ibid.*, August, 1923.

97. SAELHOF, C. C. **Papillary epithelial tumor of the orbit.** *Ibid.*, June, 1923.

98. SEEFELDER. **Orbital metastasis of a sympathetic neuroblastoma.** *Archiv f. Augenheilkunde*, xcii., p. 7.

99. STIEREN, E. **Neurofibroma of the orbit.** *American Journal of, Ophthalmology*, March, 1923.

100. THOMSON, E. S. **Orbital œdema in exopthalmic goitre.** *Ibid.* January, 1924.

101. VAN DUYSE and MARBAIX. **Metastase ethmoido-orbitaire d'hypernephrome latent.** *Archiv d'Ophtalmologie*, 1922, xxxix., No. 7.

102. WHITHAM. **Pulsating exophthalmos.** *American Journal of Ophthalmology*, February, 1923.

103. WOLLENBERG. **Exophthalmos of the left eye.** *Klinische Wochensch.*, 1922, p. 971.

ANICETO-SOLORES (81, **Hydatid cysts of the orbit**) has studied in South America hydatid cysts of the orbit induced by the hæmatogenous entrance of embryos of tænia echinococcus unilocularis, mainly in dogs, jackals, and wolves. The cysts vary in size from that of a nut, or of an egg to that of an orange. They cause pain in the orbit through pressure on the nerves and also through toxic irritation. Ptosis, pareses and paralyses of the ocular muscles follow according to the site of the hydatid. Increase of pressure leads to extreme exophthalmos. The functional disturbances in the early stage are often myopia, hypermetropia, astigmatism, paralyses of accommodation, diplopia, scotomata, and photopsias. When the optic nerve is involved a papillitis may appear early, which rarely becomes a choked disk, but often results in atrophy. Operative treatment must include a thorough removal of the inner membrane of the cyst to prevent a recurrence.

BECKER (82, **Congenital microphthalmos of both eyes, with exophthalmos and carcinoma in the left orbit**) reports the case of a child which died when 66 days old. The most interesting detail is the finding at autopsy of a congenital carcinoma of the left orbit involving the middle fossa of the skull.

The tumor of the ciliary body reported by SAELHOF (97, **Papillary epithelial tumor of orbit**) seemed to be a slowly grow-

ing benign neoplasm. The lack of mitosis and of a tendency toward infiltration confirmed this opinion. It probably originated in embryonic life and belongs to the group of blastodermata.

BEDELL (83, **Osteoma of the orbit**) removed the tumor after draining a secondary orbital abscess. It was necessary to take with it the ethmoid plate to which it was attached. It measured about 12*mm* in length. ALLING.

LATTERI (91, **Osteoma of the frontal sinus breaking into the orbit**) describes a case met with in a man 26 years old. The tumor had developed slowly in the characteristic situation at the upper, inner part of the orbit, and produced exophthalmos, diplopia and limitation of the muscular movements of the globe. Starting from the frontal sinus it had invaded the orbit. The large, very hard tumor was removed with preservation of the eye, which regained its full function in a relatively short time.

BENEDICT (85, **Tumors and cysts arising near the apex of the orbit**) prefers to remove orbital tumors through a skin incision made above the superior orbital rim. After dissecting up the periosteum of the orbital wall the tumor is located and an incision made through the periosteum nearest to its location. Tumors in the muscle cone are best removed by the Krönlein operation. Eight cases are reported, five were operated upon by the direct frontal method, three by Krönlein. The pathological report showed one sarcoma, three endothelioma, three glioma and one neurocytoma. The results in five were good, two developed brain tumor and in one it was necessary to enucleate the eyeball in removing the tumor. Having existed for about seven years the mass in the orbit had pushed the eye forward and downward and a tumor could be felt above the eyeball.

BEGLE (84, **Hæmangioma cavernosum of orbit**) performed the Krönlein operation and removed a mass which proved to be partly cystic. The final result was highly satisfactory as the position and movements of the eye were normal. The optic nerve showed temporal pallor but the acuity of vision was 20|40.

LAPERSONNE, VELTOR and PRELAT (90, **Supernumeraries in the orbit**) reports the case of a healthy 15-year-old girl who had

had a slight exophthalmos and deviation of the right eye inward from childhood. The eye could not move downward and outward; attempts to make such movements induced an oscillating nystagmus. Otherwise the eyeball presented no peculiarities. Vision was 0.3 with + 2 D spherical. The cause of the trouble was revealed by the X-ray. In the floor of the orbit was a convolution, starting from the vertical portion of the mandibula, containing about 12 supernumerary teeth, mostly resembling molars, and situated in and in front of the inferior orbital fissure. Otherwise the teeth were normal.

LUNDING-SMIDT and JENSEN (92, **A case of encephalocele in the back part of the orbit**) report the case of a two days old child born with marked protrusion of one eye. The upper lid was hypertrophic, the cornea clear, the tension was not increased, reaction to light was present, and the movements of the eyeball were limited only in a lateral direction. The eyeball could be pressed back somewhat into the orbit, but each time caused great disturbance with lusty shrieks. Enucleation of the eyeball revealed a fluctuating, transparent tumor as large as a walnut united posteriorly with the wall of the orbit and containing a serous fluid. Three months later the tumor had reformed and was well covered by the lids. Compression caused a slowing of the pulse and vomiting.

DI MARZIO'S (93, **Chloroma of the orbit**) patient was a boy 7 years old. The disease began with painful exophthalmos, first of one eye, then of the other, with progressive anæmia and fever. The tumors grew rapidly from the roof of the orbits, displacing the eyeballs, over which the upper lids were tense and greenish. Ulceration and perforation of the left cornea soon resulted from the inability of the lids to close. Cachexia increased, there was a profuse, ill smelling, greenish, purulent secretion, and the patient died at the end of three months. Autopsy revealed a large greenish tumor of elastic consistence and smooth surface which enveloped the collapsed globe, filled the left orbit and involved the cavernous sinus, sphenoid, ethmoid, maxillary sinus and the roof of the right orbit. Diagnosis, chloroma.

MEISSNER (94, **Tubercle bacilli in the orbital tissues**) says that while tuberculous changes starting from the periosteum create a typical clinical picture, the differential diagnosis of

retrobular tuberculosis of the contents of the orbit is very difficult. The only diagnosis that can usually be made is that of a retrobulbar tumor, or of inflammation. The X-rays, examinations of the accessory sinuses, Wassermann's test, and excision of bits for examination give no help. These difficulties of diagnosis were illustrated by his patient, a woman 52 years old, who had an exophthalmos which was slowly increasing. The vision was badly impaired, a central scotoma was present, a stasis of the retinal vessels and a rather prominent papilla could be observed. Krönlein's operation was performed. The temporal portion of the orbit was found to be free, but the nasal portion was occupied by a tumor which embraced the optic nerve and appeared to come posteriorly from the ethmoid. A piece of this tumor was excised, from which the diagnosis of carcinoma coming from the ethmoid was obtained. The orbit was then eviscerated. Microscopic examination of the contents of the orbit revealed typical tuberculosis with few bacilli. The infection appeared to have begun in one of the cells of the ethmoid, as indicated clinically by the central scotoma and the condition found at the first operation, in spite of the negative results of the rhinological examination, as tuberculosis of the posterior ethmoid cells cannot be diagnosed unless a secondary sinusitis is present. Because of the age of the patient it is thought that the primary seat of the disease was in the mucous membrane.

SEEFELDER (98, **Orbital metastasis of a sympathetic neuroblastoma**) found in a child 13 months old a distinct exophthalmos, some œdema of the lids, and limitation of mobility of the eyes. A blue discoloration of the lower lid was ascribed to hemorrhage. The pupils were dilated and fixed, the papilla pale. General examination revealed a tumor in the region of the pelvic hollow of the sacral bone and swollen lymphatic glands X-ray negative. The blood showed a high degree of anæmia with disturbance of the production of lymphocytes and leucocytes. Tumors appeared in the region of the temple, and cutaneous hemorrhages near the bone metastases. The diagnosis was tumor of the suprarenal capsules with metastases which was confirmed by autopsy.

STIEREN'S (99, **Neurofibroma of the orbit**) patient gave a history of increasing hypermetropia, enlarged and tortuous

veins and failing vision in one eye. Slight exophthalmos and limited motility developed. An encapsulated tumor was removed from the orbit, the operation being facilitated by cutting away about half an inch of the outer orbital rim. It proved to be a neurofibroma arising probably from the supporting tissue of the optic nerve. Single neuromata of the orbit are rare.

The 67 years old man observed by VAN DUYSE and MARBAIX (101, **Metastasis of a latent hypernephroma in the ethmoid and orbit**) suffered from nosebleed, purulent nasal secretion, headache, and a slight exophthalmos, all on the same side of the head. At the inner upper angle of the orbit a soft, tender tumor could be felt. The nasal mucous membrane was hypertrophic and bled easily. Visual functions normal, no diplopia. Transillumination showed shadows in the region of the frontal and maxillary sinuses. Mouro's operation was formed. After removal of the nasal process of the superior maxilla and a part of the nasal bone a soft, much bleeding tumor was extirpated, which had destroyed the inner wall of the orbit, the lower wall of the frontal sinus and part of the roof of the orbit. The dura was pressed back, but not involved, the maxillary sinus was filled by masses of tumor, the sphenoidal sinus was free. The operation wound healed smoothly in a few days. The X-rays revealed a tumor in the region of the kidneys which was thought to appertain to the suprarenal capsule. The histological examination of the extirpated growth showed a marked resemblance of its tissue to the structure of the suprarenal capsule. Rows of epithelial cells were separated by dilated capillaries. The cells of the alveoli were cuboidal, oval, polygonal, the protoplasm usually transparent. The cells showed all stages of degeneration. There were newly formed teleangiectatic areas with strata of connective tissue. Intraparenchymatous hemorrhages were common. The diagnosis was given as a metastasis from a latent hypernephroma.

CANGE (86, **Acute osteomyelitis of the horizontal process of the sphenoid**) reports a case of this nature met with in a girl 16 years old. The clinical picture began with an abscess of the orbit. The general condition grew worse, meningitic symptoms set in, and death followed, caused by an abscess

as large as a small hen's egg in the temporal lobe of the brain.

CANGE (87, **Giant mucocele of the frontal sinus**) reports a case met with in a man 33 years old, who had never been seriously sick and came from a healthy family. Five years before the patient had noticed a tumor about as large as a pea, painless and apparently somewhat movable, near the root of the nose. The tumor had increased in size rapidly. When he came under observation he had extreme exophthalmos, the eyeball being pushed completely out of the orbit, the cornea turned to the side, and its mobility limited, especially upwards and inwards. The papilla and its neighborhood was œdematous, the veins were distended and tortuous, arterial pulsation was present. Vision was 0.1. The general condition was excellent, Wassermann negative, urine normal. The X-ray showed perfectly normal conditions on one side, while on the other the frontal sinus and orbit had been converted into a single cavity as large as a hen's egg. As total extirpaiton was impossible the tumor was divided and emptied of its greenish, glutinous contents. The obliterated naso-frontal canal was made permeable, tamponed, and the cutaneous wound closed with sutures. The patient was discharged four weeks later after a normal recovery. Eight months later the eyeball was in the orbit, having a divergent strabismus of ten degrees, while the movements of the eyeball inward, outward and downward were limited to about ten degrees. Movement upward was difficult and by jerks. The margins of the papilla were still rather hazy, the arteries small, the veins, especially the upper ones, fuller and tortuous. Vision was 0.5. The visual field had normal limits, only at times diplopia occurred. The bacteriological examination of the contents of the cyst gave a negative result.

v. d. HOEVE (89, **The optic nerve and the accessory sinuses**) says that the ophthalmologist can find nothing in the eye to guide him to the cause of a retrobulbar neuritis. Disturbances of the light sense, if the visual field, scotomata, optic atrophy, papillitis, choked disk, and retrobulbar neuritis show us that the optic nerve is diseased, but suggest nothing as to the cause of the disease. The rhinologist cannot do much better through the recognition of diseases of the accessory sinuses.

In spite of the most careful examination he often comes to no result and must say with Mackenzie that the absence of any symptom of disease within the nose does not suffice to exclude sinusitis. The X-ray has certainly facilitated the recognition of diseases of the accessory sinuses, but in many cases it is not conclusive, particularly in mucoceles of the sphenoidal cells. One set of cases of optic nerve disease recovers immediately after the accessory sinuses have been opened, another is influenced little or not at all by such treatment. If the rhinologist finds a disease, he may at first be conservative, later wish to operate. If he finds nothing, the therapeutic procedure is difficult to determine, especially in chronic cases with slight optic nerve disturbance. Only the constant coöperation of the ophthalmologist and rhinologist, and the accurate weighing of the advantages and disadvantages of an operation can give desirable results.

WOLLENBERG (103, **Exophthalmos of the left eye**) reports a case of sudden exophthalmos in a woman 41 years old, associated with a bluish red discoloration and swelling of the lids, choked disk, and paralysis of the abducens. At the end of four weeks there was optic atrophy, and paralysis of the abducens persisted, the other symptoms had for the most part disappeared. Wollenberg thinks the cause was a retrobulbar hemorrhage due to a ruptured varix.

THOMSON (100, **Orbital œdema in exophthalmic goitre**) reports three cases of marked exophthalmos in one of which both eyes were enucleated because of breaking down of the cornea. After a review of the literature he concludes that exophthalmos is due to localized œdema in the posterior part of the orbit which for some unknown reason if sufficiently prolonged is followed by formation of connective tissue and permanent thickening. ALLING.

WHITHAM (102, **Pulsating exophthalmos**) relates seven cases, four traumatic and three spontaneous. Ligation of internal carotid was practiced in five cases and in each case the results were satisfactory. He discusses at length the statistics bearing on the various methods of treatment and concludes that cases of true carotid-cavernous sinus communication are best attacked by ligation of the internal carotid. The collateral circulation should be prepared by daily compression for

several weeks prior to the operation in which case there is little likelihood of danger. If the procedure fails the ophthalmic vein should be tied before resorting to ligation of other carotids.

MORGAN (95, **Gunshot wound of orbit**) found dilated veins and hazy nerve head in a case in which a bullet was located by the X-ray in the posterior orbit. The flattened bullet and some loose fragments were removed by the Krönlein operation with resultant vision of 20/40.

PFINGST (96, **Knife blade in the orbit**) removed a rusty knife blade from the orbit where it had remained for four months and a half without causing reaction. The eye, however, was blind and the internal and inferior recti muscles paralyzed probably from pressure of a blood clot in the orbit.

DOHERTY (88, **Formalized cartilage implants following enucleation**) advocates the introduction of spheres of beef cartilage into Tenon's capsule after enucleation. The grafts are placed in a two per cent. solution of formalin for two weeks and then transferred to normal salt solution and will keep indefinitely. He has found that the cartilage implants are well tolerated.

(*To be continued.*)

BOOK REVIEWS.

IX.—**Pathology and Bacteriology of the Eye.** By E. TREACHER COLLINS and M. STEPHEN MAYOU. Second edition. 731 pages, 4 colored plates and 306 text figures. Philadelphia, P. Blakiston's Son & Co. 1925. Price $10.00.

It is a great pleasure to welcome a second edition of Collins and Mayou's handbook. The interesting and novel classification which the authors adopted for the first edition has been retained; the book has been revised and much new material with illustrations added. The matter is presented in a most instructive and interesting way so that the book forms not a dry pathology but rather a clinical study of ocular changes based and supported by our knowledge of embryology, pathology and bacteriology. A great deal of material is presented in a truly scientific yet practical manner, written in clear and simple language, which deserves the most careful study of the advanced, as well as the beginning ophthalmologist.

A. K.

X.—**Newer Methods of Ophthalmic Plastic Surgery.** By DR. E. B. SPAETH, Major M. C., U. S. A., Washington, D. C. With a foreword by Dr. W. H. Wilmer, 257 pages, 168 illustrations. 1925. Publishers, P. Blakiston's Son & Co. Philadelphia. Price, $5.00 net.

The impetus which the war gave to plastic surgery of the face makes a book on this subject timely if for no other purpose than to collect and arrange in an accessible form the improvements in old methods and the new operations which have been developed. The author has had extensive experience in this branch of surgery not only during the war but

in the period which followed, both in war and industrial accidents.

After a general surgical introduction the various flaps, epithelial, free or pedunculated dermic, cartilage, bone and fascial are described and their application in the various lesions and deformities explained. The text is clearly expressed, copiously illustrated with drawings from recognized authorities and from personally observed cases.

The ingenuity and patience of the surgeon who practices plastic surgery are frequently taxed and as the experience of a single surgeon is often limited, a guide to the best operations from the many methods advocated is a real help. Major Spaeth's book will therefore come as a welcome and important addition to the ophthalmic surgeon's working library.

A. K.

XI.—**Refraction und Akkomodation des Menschlichen Auges (Refraction and Accommodation of the Human Eye.)** By Professor A. Siegrist, Director of the University Eye Clinic, Berne, Switzerland, 148 pages, 108 illustrations, in great part colored. Publisher, J. Springer, Berlin, 1925. Price, 18.60 marks.

This book is based on Professor Siegrist's lectures to students and physicians. It does not aim to be an exhaustive treatise and mathematical formulæ are omitted. After giving the anatomy and histology of the eye and the ophthalmoscopic appearance of the normal eye-ground, chapters are devoted to dioptics, to the optical principles of spectacles, to accommodation and to visual acuity. The refractive errors are then treated on broad clinical lines without touching upon the actual examination, either subjective or objective for refractive errors. The chapter on myopia is noteworthy, not only for its subject matter but for its illustrations. There are excellent drawings of the myopic eyeball, of the myopic eye-ground with the various normal and pathologic changes, of microscopic sections of the myopic optic nerve head, and of the changes resulting from the myopic conus. The beauty of these colored reproductions is rarely equalled in the most expensive atlas.

A. K.

XII.—**Ocular Therapeutics.** By Dr. E. Franke, Hamburg. Translated by Dr. Clarence Loeb, Chicago. 183 pages. Publishers: C. V. Mosby Co., St. Louis, 1925. Price, $3.50.

After an introduction stressing the importance of the general treatment in eye lesions, general treatment is described in tuberculosis and syphilis. Serotherapy, organotherapy, treatment with foreign proteids, ray and light therapy and electric treatment conclude the chapter. Under medical treatment will be found an alphabetical list of the drugs in general use together with their action and dosage. The use of heat and cold, baths, bath and spring cures are discussed in a final chapter. Under the heading of local treatment the author describes mechanical treatment: massage, blood letting, expression, bandaging, the application of cold and heat, light and ray treatment; and adds a list of the drugs which are to be applied locally. Electrotherapy, electrolysis, iontophoresis, galvanocautery are then taken up in turn. In the special part arranged according to the diseases of the various anatomic parts of the eye, a description is given of the remedies which are indicated and found of use.

The therapeutic field has been carefully covered by the author; the important methods of treatment are fully given, and in the selection of the remedies, a conservative yet adequate choice has been made. The work of the translator is excellent.

The author and translator are to be congratulated on the publication of this timely book. It is based on the author's large experience and will prove to be of great value to every practitioner.

A. K.

XIII.—**Myelogenetisch-Anatomische Untersuchungen Ueber Den Zentralen Abschnitt Der Sehleitung** (Myelogenetic-Anatomic Investigations on the Central Division of the Optic Radiation). By Dr. R. A. Pfeifer, Professor of Neurology, Leipzic University. 149 pages, with 119 illustrations, partly in color. Publisher: J. Springer, Berlin, 1925.

This anatomic investigation of the optic radiation is based on a thorough study of the literature and on the results of the

author's own studies with myelogenetic-anatomic methods. These have resulted in some new and original viewpoints. Our knowledge of this difficult and important subject is thereby furthered and the way to additional study facilitated.

A. K.

INDEX OF AUTHORS AND SUBJECTS.

VOL. LIV.

Accommodation, Subnormal, (Alexander Duane) 566
ADLER, FRANCIS H., and LANDIS, E. M., Studies on the Protein Contents of the Aqueous Humor. Effects of Miotics and Mydriatics, 265
Anatomy, Physiology and Pathology; Reports on progress of, 517
Aniridia, 387
Ankyloblepharon, Congenital, 194
Aqueous Humor, Studies on the Protein Contents of. Effects of Miotics and Mydriatics, (F. H. ADLER and E. M. LANDIS) 265

BLAAUW, E. E., Circumscribed Lamellar Lens Change Probably Inherited, 305
BLAKE, EUGENE M., A Collective Study of the Treatment of Congenital Hydrophthalmus, 1
Blindness, Congenital, without Obvious Cause, 393
Book Reviews, 108, 209, 417, 617
BORDLEY, JAMES, JR., The Significance of Papilledema to the Ophthalmologist, 158
British Medical Association Meeting, 318
BRUECKNER, A., "Glasses and the Eye at Rest," ("Grundzuege der Brillenlehre fuer Augenaerzte") Book Notice of, 109
BRUNER, ABRAM B., The Visual Complex, 372
BRUNS, H. DICKSON, Syphilis and Iritis, 462
BURCH, FRANK E., The Use of the Tendon Tucker—A Method of Suturing with Silk, 333

Carcinoma; Metastatic of Eyeball, with Unusual Features, 389
Cataracts, Inherited Congenital, 309
Cataract, Serum and Lens-Antigen Extract treatment for the Prevention and Cure of, (A. Edward Davis) 172
Cataract, Smith Operation for, 515
Caustic Lye: Destruction of Eye from, 387
Cavernous Atrophy and Glaucoma, 195
Cerebral Tumor, with Eye Syndrome, 508
Choroid, Bilateral Metastatic Carcinoma of the, 205
Choroid, Melano-Sarcoma of the, Followed by Phthisis Bulbi and Irido-Cyclitis in Both Eyes, (Martin Cohen) 58
Chorio-Retinitis, 383
CLARKE, ERNEST, "Errors of Accommodation and Refraction," Review of, 209
Clinical Exhibit at Westminster Eye Hospital, 200
Cocain Substitutes: Comparative Value of, Discussion on, 86
COHEN, M., Report of a Case of Melano-Sarcoma of the Choroid, Followed by Phthisis Bulbi and Irido-Cyclitis in Both Eyes, 58
COHEN, MARTIN, Report of a Case of Orbital Neurinoma, 426
COLLINS, TREACHER; and MAYOU, M. STEPHEN, "Pathology and Bacteriology of the Eye," Review of, 617
Congenital Hydrophthalmus, a Collective Study of the Treatment of, (Eugene M. Blake) 1

Conjunctiva, Amyloid Degeneration of the, (R. E. Wright) 280
Conjunctiva, Obscure Affection of the, Resembling Cases of Parinaud's Syndrome, (R. E. Wright) 423
Cornea, Conservative Operative Treatment of Large Staphylomata of the, (R. E. Wright) 55
Cornea, Epithelial Dystrophy and Recurrent Erosion of the, as Seen with the Slit-Lamp, (S. R. Gifford) 217
Couching, Plea for Reviving the Operation for, (G. Lindsay Johnson) 466
Critchett, Sir Anderson, Tribute to, 399
CURRAN, E. J., Subconjunctival Cauterization Over the Ciliary Body, with the Galvano-Cautery, to Reduce Intraocular Pressure in Advanced Glaucoma; Preliminary Report, 321

Dacryoadenitis Acute, from Local Infection, (M. L. Foster) 430
Dacryocystitis, Inflammatory Swellings Simulating, (John Green) 68
Dacryops, (Otto Landman) 592
DAVIS, A. EDWARD, Serum and Lens-Antigen Extract Treatment for the Prevention and Cure of Cataract, 172
DE BLASCOVICS, L., On Results of Turning of the Tarsus, 38
Descemet's Membrane, Excrescences of Central Area of, (Daniel B. Kirby) 588, 598
DE SCHWEINITZ, G. E., "Diseases of the Eye," Review of, 210
DUANE, ALEXANDER, Projection and Double Vision, New View Points, 233
DUANE, ALEXANDER, Subnormal Accommodation, 566

EDMUND, CARSTEN and MOELLER, H. ULRIK, Vision in Light of Reduced Intensity, 531
Endocrine Origin, Ophthalmic Disorders of, in Women, (O. R. Lourie) 469
English-Speaking Ophthalmological Societies, Convention of, 213
Exophthalmos, Pulsating, 509
Exudate, a Massive, 508
Eye, The, In Relations to General Diseases and Poisons, Reports on Progress in, 94
Eye Accidents, Industrial, 309
Eye Examinations, Periodic, 309
Eyeball, Dislocation of, without Demonstrable Fracture of the Orbit or Rupture of the Sclera, (S. P. Oast) 191
Eyelids, Syphilitic Gumma of the, (Edward B. Heckel and Lawrence G. Beinhauer), 352
Eyes in Intra-uterine Life, 194

Focal Illumination with a Combined Corneal Magnifier, 390
FOSTER, MATTHIAS LANCKTON, Acute Dacryoadenitis, Caused by Local Infection, 430
FRANKE, E., "Ocular Therapeutics," Review of, 619
FRIEDENWALD, HARRY, Vascular Lesions of the Retina in Young Persons, 111
FRIEDENWALD, H., and FRIEDENWALD, J. S., Melanosis of the Lids, Conjunctiva, and Sclera, with Wart-Like Growths on the Iris, 51
FRIEDENWALD, JONAS S., Coloboma of the Mesodermal Layer of the Iris, 349
Fundus Oculi in General Medicine Discussion on the, 604

GIFFORD, HAROLD, Miscellaneous Notes on Glaucoma, 337
GIFFORD, S. R., Epithelial Dystrophy and Recurrent Erosion of the Cornea as Seen with the Slit-Lamp, 217
Glaucoma, Subconjunctival Cauterization of the Sclera Over the Ciliary Body with Galvano Cautery to Reduce Intraocular Pressure, in Advanced, (E. J. Curran), 321
Glaucoma, Knapp Adrenalin Mydriasis Reaction in Direct Descendants of Patients with Primary, (M. J. Schoenberg) 282
Glaucoma, Pathogenesis of, (F. H. Verhoeff) 20
Glioma and X-ray Treatment, 406
Gliosis Retinæ, Origin of, in Cases of Staphyloma Corneæ, (Peter S. Soudakoff) 288
Gonioscope, 207

GREEN, JOHN, Inflammatory Swellings Simulating Dacryocystitis, 68
Gumma, Retrobulbar, 313

HAESSLER, F. HERBERT, Heterochromia Iridis, 298
HECKEL, EDWARD B., and BEINHAUER, LAWRENCE, G., Syphilitic Gumma of the Eyelid. Case Reports, 352
HELMHOLTZ, "Physiological Optics," Review of, 211
Hemianopia, Bi-temporal, from Skull Fracture, 393
Heterophoria, Non-Surgical Treatment of, in Ametropia, (Herbert Wright Wootton) 440
HIRSCHBERG, JULIUS, Obituary Notice of, 418
HORRAX, GILBERT, The Significance of Papilledema to the Neurological Surgeon, 130
Hydrophthalmus, A Collective Study of the Treatment of Congenital, (Eugene M. Blake) 1
Hypermetropia, Excessive, 384

Implantation Cyst, 194
Irido-Cyclitis, A Case of Tubercular, with Fatty Degeneration and Infiltration of the Anterior Segment of the Eye, (Otto Landman) 261
Irido-Cyclitis, Intradermal Test with Uveal Pigment in Traumatic, (Arnold Knapp) 252
Iris, Heterochromia of, (F. Herbert Haessler) 298
Iris, Coloboma of the Mesodermal Layer of the, (Jonas S. Friedenwald) 349
Iris, Traumatic Cyst of the, with Glaucoma, 91

JOHNSON, G. LINDSAY, A Plea for Reviving the Operation for Couching, 466

Keratitis Punctata Superficialis, (A. W. Stirling) 432
Keratitis, Tubercular, in a Cat, 502
Kirby, Daniel B., Excrescences of the Central Area of Descemet's Membrane, 588
KNAPP, ARNOLD, Intradermal Test with Uveal Pigment in Traumatic Irido-Cyclitis, 252

Lachrymal Needle, 92
Lacrymal Sac, Operations on, 510
LANDMAN, OTTO, A Case of Tubercular Irido-Cyclitis with Fatty Degeneration and Infiltration of the Anterior Segment of the Eye, 261
LANDMAN, OTTO, Dacryops, Three Cases of, 592
LANG, BASIL, "The Routine Examination of the Eye," Notice of, 417
Lens, Lamellar Changes of the, (E. E. Blaauw) 305
LEVINSOHN, G., Note on the Genesis of Myopia, 434
Lids, Conjunctiva, and Sclera, Melanosis of the, with Wartlike Growths on the Iris, (H. Friedenwald and Jonas S. Friedenwald) 51
Lipemia Retinalis, 205
LOURIE, O. R., Ophthalmic Disorders of Endocrine Origin in Women, 469
LOWENSTEIN, ARNOLD, "Die Tuberculose des Auges," ("Tuberkulosis of the Eye"), Review of, 210
Lupus, Bilateral Ectropion of the Lower Lids, from, 90

Macula, Traumatic Hole in, 408
Macular, Disease, 195
Macular Vision, Percentage Evaluation of, (A. C. Snell, and Scott Sterling) 443
Man and Animals, Eye Diseases, Common to, 503
MAY, C. H., "Manual of the Diseases of the Eye" Review of, 211
Medullated Fibres; Extensive, 388
Miners' Nystagmus, *see also* elsewhere, 402
Myopia, On the Genesis of, (G. Levinsohn) 434

Neuro-Fibroma, Plexiform, 388
Neurofibromatosis, with Buphthalmus, (Alfred Wiener) 481
New York Academy of Medicine, Meeting of Ophthalmological Section of, 90, 205, 308, 383, 489, 598
Nocardia, (Streptothrix) Infection from, (F. E. Woodruff) 11
Notice, 213, 318

Nuclear Atrophy, with Bilateral Facial Paralysis, 406
Nystagmus, Miners', 196

OAST, S. P., Pronounced Traumatic Dislocation of Eyeball without Demonstrable Fracture of Orbit, or Rupture of the Sclera, 191
Ophthalmology, Reports on the Progress of, 94, 409, 517, 608
Optic Atrophy, Blindness Due to, 308
Optic Atrophy, The Treatment of, (E. L. Zimmermann) 545
Optic Canal, Value of X-rays of, 600
Optic Disc, Hole in the, 74
Optic Nerve, Tumor of, 400
Orbit, Inflammatory Pseudo-Tumors of the, 397
Orbit, Metastatic Carcinoma of the, 313
Orbit, Recovery from Pseudo-Tumor of, 399
Orbit and Accessory Sinuses, Reports on Diseases of the, 608
Orbital Neurinoma, Report of a Case of, (Martin Cohen) 426

Papilledema in Oto-Laryngology, (Leon, E. White) 142
Papilledema, Significance of, to the Neurological Surgeon, (G. Horrax) 130
Papilledema, Significance of, to the Ophthalmologist, (James Bordley, Jr.) 158
Papillitis, Abrupt Monocular Blindness, Resulting in Complete Optic Atrophy, During Encephalitis. X-ray of Optic Canals (Clifford B. Walker) 165
Paralysis, Diplopia in, 206
PFEIFER, R. A., "Myelogenetisch-Anatomische Untersuchungen Ueber den Zentralen Abschnitt der Sehleitung," ("Myelogenetic-Anatomic Investigations on the Central Division of the Optic Radiation"), Review of, 619
Phacœrisis, Findings after, 391
Projection and Double Vision, New View Points, (A. Duane) 233

Remedies and Instruments, Reports on, 409
Retina, Angioma of the, 401
Retina, Angiomatosis of the, (Hunter W. Scarlett) 183
Retina, Detachment with Hole in Retina, 407
Retina, Physiology of, in Perimetry and Campimetry, 384
Retina, Vascular Lesions of the, in Young Persons, (H. Friedenwald) 111
Retinitis Hemorrhagica, Unilateral, 93
ROMAINS, JULES, "Eyeless Sight," Review of, 108
Royal Society of Medicine, Reports of Transactions of the Section of Ophthalmology of, 74, 194, 393, 502

Sarcoma, Epibulbar, 510
SCARLETT, HUNTER W., Angiomatosis of the Retina, 183
SCHOENBERG, MARK J., The Knapp Adrenalin Mydriasis Reaction in Direct Descendants of Patients with Primary Glaucoma, 282
SIEGRIST, A., "Refraction and Akkomodation des Menschlichen Auges," ("Refraction and Accommodation of the Human Eye") Review of, 618
Slit-Lamp Examinations, 598
SNELL, ALBERT C., and STERLING, SCOTT, The Percentage Evaluation of Macular Vision, 443
Snowball Opacities, 502
SOUDAKOFF, PETER S., The Origin of Gliosis Retinæ in Cases of Staphyloma Corneæ, 288
SPAETH, E. B., "Newer Methods of Ophthalmic Plastic Surgery," Review of, 617
Spectacles and Magnifiers, Telescopic, 314
STERLING, SCOTT, Percentage of Evaluation of Macular Vision, (with A. C. Snell) 443
STIRLING, A. W., A Case of "Keratitis Punctata Superficialis," 432
Syphilis and Iritis, (H. Dickson Bruns) 462

Tarsus, Results of Turning the, (L. de Blascovics) 38
Tendon-Tucker, Use of, and a Method of Suturing with Silk, (Frank E. Burch), 333
Tints and Their Values, 74

Ultra-violet Radiation, Susceptibility of Nocturnal Animals to, 394

VERHOEFF, F. H., The Pathogenesis of Glaucoma, 20
Vertigo, Discussion on, 81
Vision, in Light of Reduced Intensity, (Carsten Edmŭnd, and H. Ulrik Moeller) 531
Visual Complex, (Abram B. Bruner) 352
Visual Field, Homonymous Quadrant Defects of, 313
Visual Field, New Anatomical Notation for the, 602
Vomiting, Cyclical with Ptosis, 406

WALKER, CLARENCE, B., Abrupt Monocular Blindness, Resulting in Complete Optic Atrophy, During Encephalitis, with Double Papillitis. X-ray of Optic Canals, 165
WHITE, Leon, E. Papilledema in Oto-Laryngology, 142
WIENER, ALFRED, Case of Neurofibromatosis with Buphthalmus, 481
WOODRUFF, F. E., Nocardia (Streptothrix) Infections, 11
WOOTTON, HERBERT WRIGHT, The Non-Surgical Treatment of Heterophoria, in Ametropia, 440
WRIGHT, R. E., A Case of Amyloid Degeneration of the Conjunctiva, 280
WRIGHT, R. E., Conservative Operative Treatment of Large Staphylomata of the Cornea, 55
WRIGHT, R. E., Obscure Affection of the Conjunctiva, Resembling Cases of Parinaud's Syndrome, 423

ZIMMERMANN, E. L., The Treatment of Tabetic Optic Atrophy, 545

Engraving and Stationery

A large number of people who reside at a distance from the principal cities, not understanding the facility and accuracy with which orders for Engraving and Stationery can be filled through the mails, are obliged to content themselves with a very inferior grade of Stationery, and of Engraving and Printing, when, by sending their orders to headquarters, they can have the same executed at moderate prices and in a thoroughly artistic manner. In the extensive Stationery Department of Messrs. G. P. PUTNAM'S SONS, Original Designs for Monograms, Crests, and Coats-of-Arms are furnished for stamping on note and letter paper. Visiting Cards, Wedding and Reception Invitations are engraved and printed in the correct style, while their exceptional facilities enable PUTNAMS to execute all such work at the lowest prices that are consistent with a high standard of excellence.

Their stock of Fine Writing Papers is extensive, and contains all the latest novelties, many of the same being specialties manufactured exclusively for them. PUTNAMS are now conducting an extensive trade through the mails, and are in a position to supply the wants of their customers promptly, intelligently, and economically. They furnish estimates and samples for every description of engraving and printing.

Putnams

2, 4, and 6 West 45th St., New York **24 Bedford St., Strand, London**